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PARTICULATE EXPOSURE DURING THE PERSIAN GULF WAR

FINAL

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I. EXECUTIVE SUMMARY

Troop exposure to air pollution and respirable particulate matter (PM_{10}) during the Gulf War (1990–1991) is of concern because of potential health effects. This report examines breathing zone particulate pollution of military personnel during the Gulf War. While both suspended and respirable particulate contaminants were determined to be at elevated levels, the focus of this report was to examine the respirable silica fraction. Estimates of respirable silica and soot concentrations are made from troop exposure monitoring data. These calculations are then compared with widely accepted U.S. exposure guidelines and occupational exposure studies.

Dermal exposures to silica dust are also considered in this report. Silica dusts are associated with specific types of dermatitis or skin inflammation. A dry, dusty environment can promote minor skin reactions. These irritations are not expected to initiate long-term chronic skin disorders, however. Long-term dermal exposures to silica dusts are associated with connective tissue diseases with a potential to produce progressive systemic scleroderma, which commonly affects hand and facial tissue. Systemic scleroderma is diagnosed when symptoms are widespread to include the entire body. Credible evidence exists between scleroderma, occupational settings, and increased risk of silicosis; however, no increased evidence of scleroderma has been identified at low crystalline silica exposure levels. Gulf War soldiers were not exposed to silica dust greater than an adjusted cumulative three-year duration or at levels approaching occupational settings where silicosis is present. Therefore, health risks resulting from dermal exposure are negligible.

Particulate matter is the generic term applied to a broad class of chemically and physically diverse substances that exist as discrete particles (liquid droplets or solids) over a range of particle sizes. In the Gulf War environment, airborne particulate matter was chemically heterogeneous. Some originated from combustion products associated with the more than 600 oil-well fires that were set during the conflict, in addition to natural sources such as soil, dust, sea spray, fires, plant pollens, and assorted human activities.

Ambient particulates (i.e., particulate levels present in the general environment without regard to their specific sources) have been associated with increases in mortality and risks of respiratory disease in the general population. Further, exposures to higher concentrations of ambient particulates have been associated with changes in lung function, tissue damage, and changes in the respiratory system's defense mechanisms. Occupational exposures to respirable particulates and silica dusts have produced the same types of changes in lung tissue and function as ambient exposures. These changes, however, may be more pronounced and severe. There is a large quantity of literature describing both human occupational and laboratory animal respiratory exposures to particulates.

The U.S. Army Environmental Hygiene Agency (USAEHA) *Kuwait Oil Fire Health Risk Assessment, Final Report* (1994) indicated that the U.S. Environmental

Protection Agency (EPA) National Ambient Air Quality Standards (NAAQS) for PM_{10} were frequently exceeded during the Gulf War. Although science is uncertain as to the specific components in PM_{10} that may cause harm, this report examined scientific literature and particulate data developed during the Gulf War for a variety of dust components detected to provide as accurately as possible scientific perspective about their toxicological significance. The literature review also indicated negligible to nonexistent health risk from other inhaled particulate material. However, because silica is widespread in desert dust and highly toxic in certain forms, the emphasis of this report is on potential health effects of respirable silica exposure to troops.

Silica is a significant respirable particulate found in the Gulf Region environment. This toxic substance has the potential to produce disease in military and allied personnel. Studies in laboratory animals and humans indicate that fibrogenic and silicotic lung lesions result from the inhalation of crystalline silica. Lung cancer is a secondary concern to the formation of these lesions. Epidemiological studies support this secondary carcinogenic risk and identify a small increased relative cancer risk in workers exposed over many years to crystalline silica.

The risk of adverse health effects arising from prolonged or chronic exposures to respirable silica (7–45 years) begins to occur at exposure levels above 1 $mg/m^3 \times$ year and at dosages of total respirable silica above 2,300 mg. Chronic health effects, including chronic silicosis and chronic obstructive pulmonary disease (COPD), are prominent as exposure levels reach 2 $mg/m^3 \times$ year and dosages reach greater than 4,000 mg. If cumulative exposures to respirable silica occur seven years or less after the initial exposure and exceed 3 $mg/m^3 \times$ year, there is a risk of acute health effects. This equates to a minimum range of total dose exposure of 6,500–10,000 mg (see Tab B: Figures 6–9).

Particulate matter sampling conducted in Kuwait in 1991 by USAEHA indicated that particulates less than the PM_{10} size range contained an average of 4.3% and a maximum of 6.5% silica. This is a typical content for silica in regional desert dusts. A no observed adverse effect level (NOAEL) from respirable silica dust inhaled over a lifetime (average 70 years) would be less than or equal to 1 $mg/m^3 \times$ year, or a total dose exposure of about 3,000 mg. Occupational studies indicate silica-induced disease can be caused by lower dosages of inhaled silica (e.g., 2,300 mg). However, these levels were derived from occupational exposure settings and shorter periods (less than 45 years), resulting in the 1 $mg/m^3 \times$ year cumulative exposure NOAEL to be exceeded. A discussion on the development of the NOAEL can be found in Section V, Part A (for silica) and Section VI, Part E (for soot).

Gulf War soldiers were exposed round-the-clock to silica concentrations and PM_{10}. Areas occupied by Gulf War soldiers totaled 880,000 square miles. Because continuous air sampling throughout the Gulf War Theater was impossible, USAEHA ambient air sampling sites were selected for the highest troop concentrations and long-term troop positions. Sampling sites were generally positioned downwind from oil fire locations. This sampling strategy represents a conservative, worst-case scenario for average and maximum Gulf War personnel particulate exposure. Based on this evaluation, the scientific literature, and Gulf Region exposure data, the estimated long-term risk of adverse health effects from inhaled silica is below levels of concern.

The average cumulative exposure for Gulf War soldiers to respirable silica was roughly 1/100th–1/50th of the 1 mg/m^3 × year NOAEL cumulative exposure level. Total accumulated dose was also in this range. Adverse health effects are not expected given that the average cumulative exposure to silica is below current Occupational Safety and Health Administration (OSHA) and National Institute for Occupational Safety and Health (NIOSH) workplace limits. In addition, exposure analysis shows typical levels below those that cause health concerns for soldiers redeployed in the Gulf or similar regions of the world.

U.S. military personnel will likely be deployed in regions and situations where exposure to particulate matter is a potential health concern. Findings and conclusions in this retrospective exposure study provide planning information beneficial to future military operations.

II. INTRODUCTION

A. Background

Troop exposure to air pollution and respirable dust particles below 10 microns in aerodynamic diameter (PM_{10}) during the Gulf War is of concern because of the potential health effects. Several military and civilian activities transpired during this conflict that contributed to the air pollution in the Gulf Region, including the movement of troops and supplies, the opening and use of alternative housing, and other operations unique to the hostile activities that occurred in the region. In addition, DOD military and civilian personnel were exposed to atmospheric contaminants from more than 600 burning and damaged oil wells.

Ambient particulates are associated with increases in mortality and risks of respiratory disease in the U.S. population. Further, exposures to higher concentrations of ambient particulates have been associated with changes in lung function, damage to lung tissue, and altered respiratory defense mechanisms. Occupational exposures to respirable particulates and silica have produced similar changes in lung tissue and functions. Depending on the extent, however, these exposure changes may be more pronounced and severe. A large quantity of literature describes human ambient, occupational, and laboratory animal exposure to particulates. Based on this literature, U.S. federal agencies have developed guidelines for particulate exposure assessment.

It is determined that an ambient silica exposure in PM_{10} between 3 μg/m^3 and 8 μg/m^3 (U.S. range for a continual time-weighted average [TWA]) would be the equivalent of 8.4 μg/m^3 and 22.4 μg/m^3 for an occupational level. Such exposure levels would result in 0.6–1.6 mg/m^3 × year of cumulative silica exposure for a 70-year lifetime and a 0.03%–2.4% upper bound risk of developing silicosis. This equates to a risk of

0.3% at 1 mg/m^3 × year cumulative exposure (continually breathing 0.005 mg/m^3 of silica in PM_{10} for 70 years).[1]

B. Purpose of Investigation

The focus of this report is the ground level particulate pollution in the breathing zone of military personnel during the conflict. This report examines respirable silica and soot particulate concentrations from monitoring data and estimates troop exposures. These estimates are then compared with widely accepted exposure guidelines for U.S. silica and carbon black (soot). Based on previous Persian Gulf Region ambient air monitoring studies and an emphasis on respirable silica, this report characterizes potential health risks to Gulf War veterans from particulate exposure. Risk is the probability of adverse impacts to individuals, populations, or ecosystems from exposure to toxic substances or hazardous conditions. The purpose of risk analysis and this report is to help audiences make informed and effective decisions about the potential health of Gulf War soldiers.[2]

As with most desert environments, blowing and drifting sand are important sources of respirable particulates. The ambient air may also contain smog, combustion products, and various other respirable dusts from military operations and miscellaneous activities. Further, there will be organic-based matter from wildlife and human activities. These contaminants contribute to both air pollution and possible human health effects. However, because silica is a significant toxic component of desert dust, the emphasis of this report is on the potential health effects of respirable silica exposure in troops.

C. Organization of Report

Emphasizing the respirable silica component, this report describes methods used to estimate particulate exposure, evaluate ambient monitoring data for the Gulf Region, and summarize literature review results for potential health effects of particulate exposure. Finally, comparisons are made with U.S. federal guidelines and current occupational silica exposure studies. These analyses and conclusions focus potential understanding of human health impacts from respirable silica exposure in Gulf War military personnel.

III. METHODOLOGY

The exposure assessment methodology used in this report was based on a human population monitoring study[3] and on the results from a scientific review of environmental

[1] U.S. EPA/Office of Research and Development: "Ambient Levels and Noncancer Health Effects of Inhaled Crystalline and Amorphous Silica: Health Issue Assessment" (EPA/600/R–95/115). November 1996. pp. 7-1–7-6.

[2] Louvar, J.F., & Louvar, B.D.: "Health and Environmental Risk Analysis: Fundamentals with Applications." Prentice Hall PTR Environmental Management and Engineering Series, Vol. 2 (1998). pp. 1-22 (1998).

[3] U.S. Army: "Final, Kuwait Oil Fire Health Risk Assessment." U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD, 1994.

and occupational health effects literature. The authors conducted a literature search of the major scientific databases, consulted OSHA and EPA occupational and environmental health effects literature, incorporated information from a NIOSH report on silica health effects, and compared these findings to exposure and health effects studies conducted on the Gulf War population by the DOD. Relevant exposure and health effects studies of human populations in the Middle East were also reviewed. In total, a database of 154 articles consisting of thousands of pages of information related to silica exposure, health and risk assessment was constructed and reviewed for this report. In addition, the authors conducted interviews with members of the U.S. Army Center for Health Promotion & Preventive Medicine (USACHPPM), formerly known as USAEHA, and other associated groups to assist in reviewing DOD documents they helped prepare.

The human exposure assessment used in this report is based on a standard methodology described in the National Academy of Sciences (NAS) report, *Human Exposure Assessment for Airborne Pollutants: Advances and Opportunities*.[4] This methodology is widely used by federal agencies such as EPA and OSHA.[5] It allows for the comparison of empirical occupational exposure and resultant health effects data to develop a total human exposure scenario (24 hours/7 days per week).

This methodology estimates cumulative exposure using the formula $\Delta E = C \times \Delta t$, where ΔE is the exposure of a person to air contaminants of a particular concentration (C) for a specific time period (Δt). Cumulative exposure is measured in units of concentration vs. time: in this report, $mg/m^3 \times$ year.[6]

Silica is the main respirable particulate health risk agent from the USAEHA 1991 Gulf War veterans monitoring data. The total dose for inhaled silica particulates (or PM_{10}) is estimated by multiplying cumulative exposure values by the inhalation rates. This calculation assumes that the body absorbs all the inhaled particulate matter and that soldiers have increased inhalation rates, higher metabolism, and are more physically active than the general public. These factors increase the conservatism of this health risk estimate and present a realistic exposure assessment.

An inhalation rate of 24 m^3/day of air (or 1 m^3/hour) was used in this assessment. This rate is slightly higher than 20 m^3/day suggested for adults.[7] Occupational exposures for lifetime durations normally account for 46 weeks of exposure rather than 52 weeks. Our comparison assumes a direct conversion from a standard workweek to a 24-hour/7-day exposure scenario (i.e., continuous exposure) for Gulf War soldiers. An occupationally derived time equivalent of 1.76 years was used to enable comparison between Gulf War personnel exposures and researched occupational levels. The formula used to derive this number assumes that occupational exposures are 365 days per year, 52

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[4] National Academy of Sciences: "Human Exposure Assessment for Airborne Pollutants: Advances and Opportunities." National Academy Press, 1991. pp. 17-206.

[5] U.S. EPA: "National Ambient Air Quality Standards for Particulate Matter; Final Rule" (40 CFR 50). Washington, DC: U.S. Government Printing Office, Office of the Federal Register, 1997.

[6] National Academy of Sciences: "Human Exposure Assessment for Airborne Pollutants: Advances and Opportunities." National Academy Press, 1991. pp. 39-40.

[7] Klassen, O.A., Doull, J, & Amdur, M.O.: *Casarett and Doull's Toxicology: The Basic Science of Poisons, Fifth Edition*. The McGraw-Hill Companies, Inc., 1996. pp. 75-88.

weeks per year. OSHA considers occupational exposures to be 250 days and 46 weeks per year. This is a conservative assumption, however, possibly overestimating exposure, assuming weekend rest and no vacation for a deployed Gulf War soldier.

$$50\,{}^{\mu g}/_{m^3} \times 10\% = 5\,{}^{\mu g}/_{m^3}$$
$$5\,{}^{\mu g}/_{m^3} \times 10^{-3} = 0.005\,{}^{mg}/_{m^3}$$
$$0.005\,{}^{mg}/_{m^3} \times 70\,years = 0.35\,{}^{mg}/_{m^3}\,years$$
$$0.35\,{}^{mg}/_{m^3}\,years \times \left({}^{7days}/_{5days} \times {}^{20hours}/_{10hours}\right) = 0.98\,{}^{mg}/_{m^3}$$

The respirable silica NOAEL is less than or equal to 1 mg/m^3 × year, or around 3,000 mg of total respirable silica dust inhaled over a 70-year lifetime. Current data indicate a 10% or less silica fraction for ambient PM_{10}.[8] A 10% silica fraction of EPA's 50 µg/m^3 annual PM_{10} NAAQS equals 5 µg/m^3. Therefore, a 5 µg/m^3 silica exposure for a 70-year lifetime provides an equivalent 45-year cumulative occupational exposure of 0.98 mg/m^3 or slightly less than 1 mg/m^3 × year (see adjoining formulas).

$$1.76\,years = \frac{168\,{}^{hours}/_{week}}{40\,{}^{hours}/_{week}} \times \frac{153\,days}{365\,{}^{days}/_{year}}$$

$$Cumulative Exposure = \left({}^{mg}/_{m^3}\right) \times 1.76\,years$$

The risk of chronic health effects from silica exposure of 5 to 45 years begins to occur at levels above 1 mg/m^3 × year and/or total particulate doses above 2,300 mg. Chronic health effects become more prominent at cumulative exposure levels of 2 mg/m^3 × year or dosages above 4,000 mg. There is a risk of acute or short-term health effects from respirable silica occurring seven years or less after the initial exposure if cumulative exposures exceed 3 mg/m^3 × year and exceed a minimum dose of 10,000 mg. Occupational studies indicate that silica-induced disease appears at lower dosages of inhaled silica (2,300 mg) and shorter periods (less than 45 years). Differences in setting and exposure duration cause the 1 mg/m^3 × year cumulative exposure NOAEL to be exceeded.

A value was derived to enable comparison between Gulf War soldier exposures and researched occupational levels. The average Gulf War soldier experienced 153-field day exposure duration. This equals 1.76 years of occupational exposure (see formulas).

Cumulative exposures of Gulf War soldiers between 1/20th to 1/50th the NOAEL, using 24-hour respirable silica dust measurements, were attained at King Khalid City, Khobar Towers, Riyadh, Jubayl, Ahmadi, Camp Thunderock, and the Armed Forces Military Hospital in Kuwait City sampling sites. An exception was found at the U.S. Embassy in Kuwait City. Cumulative exposure at this sample site was 1/10th the NOAEL (see Figures 1 and 2).[9]

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[8] Gift, J.S., & Faust, R.A.: "Noncancer Inhalation Toxicology of Crystalline Silica: Exposure-Response Assessment." *Journal of Exposure Analysis and Environmental Epidemiology* 1997, Vol. 7, No. 3:345-358.

[9] U.S. Army: "Final, Kuwait Oil Fire Health Risk Assessment — Appendix B." U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD, 1994.

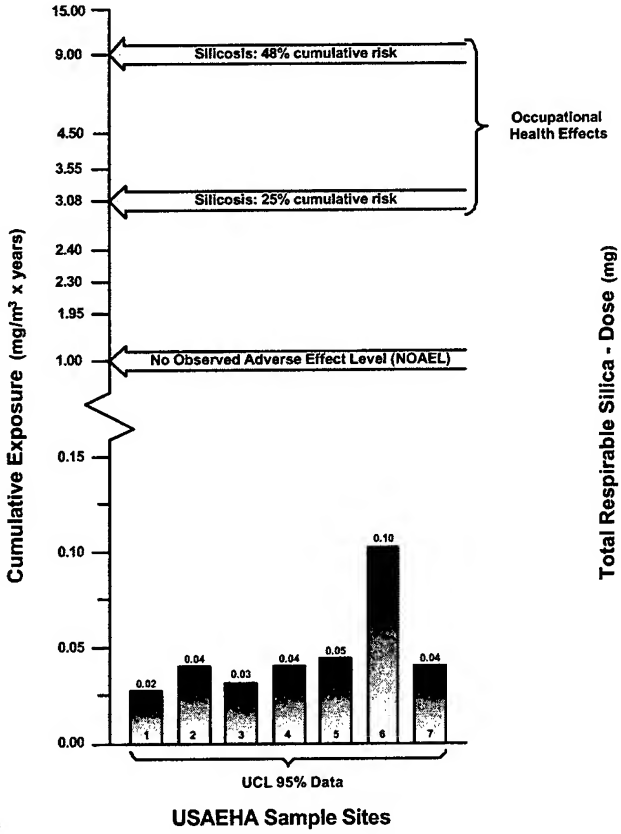

Figure 1. Cumulative exposure (mg/m^3 × years) levels are calculated from USAEHA air analysis data collected at the following sample sites: 1-KKMC, 2-Khobar, 3-Riyadh (Eskan), 4-Jubayl, 5-Camp Thunderock, 6-U.S. Embassy, 7-Military Hospital.

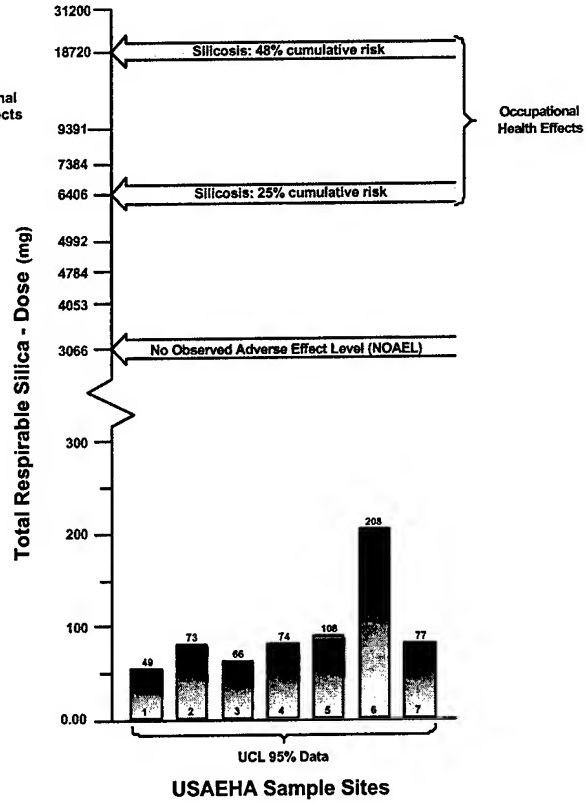

Figure 2. Respirable silica exposure levels for Gulf War veterans are identified and compared with occupational health effects studies. Total respirable silica dose (mg) levels are calculated from USAEHA air analysis data collected at the following sample sites: 1-KKMC; 2-Khobar; 3-Riyadh (Eskan); 4-Jubayl; 5-Camp Thunderock; 6-U.S..Embassy; 7-Military Hospital.

Other assumptions used to develop the exposure estimates are listed below:

- All exposures were continuous at a single concentration.
- Indirect environmental monitoring or microenvironment concentrations represent actual exposures.
- Available personnel monitoring data represent actual exposures.
- A 168 hour/week (24 hours/7 days/week) estimate was used for Gulf War soldier cumulative exposure and dosage estimates.
- A 40-hour/week (8 hours/5 days/week) occupational exposure scenario was used as a comparison estimate.
- Total exposure time for Gulf War soldiers is measured in average field days, which was determined from service records to be 153 days.[10]
- Respirable silica particulates are alpha-quartz particles 10 microns or less in size.

[10] U.S. Army CHPPM and U.S. Air Force Armstrong Laboratory: "Environmental Assessment: Eskan Village, Kingdom of Saudi Arabia," 1996.

- Analysis of the USAEHA report identifies the Gulf Region respirable particulate matter to contain an average of 4.3% and a maximum of 6.5% silica content.
- The silicon-rich fraction is expected to be 100% alpha-quartz.
- All soldiers in the field respond similarly to healthy occupational populations.
- The inhalation rate would be similar for all individuals.

Gulf War exposure estimates derived from the NAS methodology are compared with U.S. federal occupational exposure guidelines. Occupational guidelines are used for comparison purposes. There are considerable published data for occupational exposures to silica in industrial and other commercial activities. Further, exposure estimates are compared with dose–response relationships in the scientific literature where NOAELs have been determined. The risk of silicosis associated with ambient levels of respirable silica is estimated by first adjusting from 8-hour occupational exposure equivalents. This method is consistent with EPA dose–response assessments and uses the same NOAEL.[11]

IV. PHYSICAL PROPERTIES

A. Respirable Particulates

Respirable particulates may be composed of a wide range of substances equal to or less than 10 μm. Particulate matter is a generic term for many different types of particles that are both chemically and physically different. These particles may contain various metals, organic material, inert solids, and reactive chemicals. Sand in Saudi Arabia and Kuwait is composed of quartz with calcium carbonate, calcium magnesium carbonate, and silicates of aluminum.[12] The most common large particulate (greater than 20 microns) in the desert is coarse silica, or sand. Smaller respirable particles (less than 10 microns) are lower in silica and higher in clay content.[13] It is notable that commonly used PM_{10} sampling apparatus may overestimate the particulate mass actually reaching the deep lung. The respirable particulate exposure, therefore, is likely overestimated.[14]

Crystalline silica is the primary constituent of quartz and a dioxide of the element silicon. Silicon is the second most abundant chemical element in the earth's crust. Alpha-quartz is the most stable form of crystalline silica in the environment. In fact, the overwhelming majority of natural crystalline silica exists as alpha-quartz. Most

[11] U.S. EPA/Office of Research and Development: "Ambient Levels and Noncancer Health Effects of Inhaled Crystalline and Amorphous Silica: Health Issue Assessment" (EPA/600/R–95/115). November 1996. pp. 7-1–7-6.

[12] Richards, A.L., Hyams, K.C., Watts, D.M., Roxmajzl, P.J., Woody, J.N., & Merrell, B.R.: "Respiratory Disease among Military Personnel in Saudi Arabia during Operation Desert Shield." *Am. J. Pub. Health 83(9):*1326-1329 (Sept. 1993).

[13] Gomes, L., & Bergametti, G.: "Submicron Desert Dusts: A Sandblasting Process." *J. Geophysical Research 95(D9)13:*927-935 (August 1990).

[14] Lippman, M., Xiong, J., & Wei, L.: "Development of a Continuous Monitoring System for PM_{10} and Components of $PM_{2.5}$." *Appl. Occup. Environ. Hyg. 15(1):*57-67 (Jan. 2000).

laboratory animal studies in the published literature were conducted using alpha-quartz.[15] Environmental silica in air comes from both natural and anthropogenic (e.g., industrial, agricultural, construction, and transportation) activities.

B. Biological Reactivity

Biological reactivity can be defined as the interaction of a substance with living tissues and cells. The structure, size, composition, surface, and dissolution properties of silica are among the factors most likely to affect its biological reactivity.[16] Laboratory studies indicate that manufactured silica, amorphous silica, crystalline silica, and silica with trace elemental compositions differ with respect to biological reactivity. For example, the presence of iron on the surface of silica particles enhances the ability to form free radicals and, therefore, damage tissues. Aluminum on the surface may have a protective effect or act as a prophylactic agent to prevent cellular damage. Other materials, such as petroleum solvents and carbon, may increase the toxicity of silica. Differences depend on factors such as increased solubility and membrane transport.

Whether or not the silica has been freshly fractured is the factor with the strongest influence on biological reactivity. Freshly fractured silica is much more toxic than unfractured or aged silica. It is most likely found in activities involving sandblasting, rock drilling, tunneling, and silica milling operations (i.e., operations in which silica particles are crushed or sheared under conditions of high pressure).[17] This was first suspected to be an important factor as early as 1945,[18] and recently was confirmed experimentally.[19,20] Although forces and pressures necessary to fracture silica are unlikely in ambient environmental or natural settings, it is unknown at this time how much silica was freshly fractured in the Persian Gulf region from mechanical disturbances and weather conditions such as sandstorms.

Since freshly fractured silica is produced during grinding, polishing, and cutting, it is highly likely that most occupational exposure arises from man-made operations where silica is freshly fractured. Absent quantitative data, it is probable that Gulf War soldier silica exposure was primarily of aged natural origin and unlikely to have a high freshly fractured content. This uncertainty about how much silica was freshly fractured

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[15] International Agency for Research on Cancer: *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Vol. 68: Silica, Some Silicates, Coal Dust and Para-aramid Fibrils*. World Health Organization, IARC Press, Geneva, Switzerland, 1997. pp. 41-85.

[16] Guthrie, G.D., & Heaney, P.J.: "Mineralogical Characteristics of Silica Polymorphs in Relation to their Biological Activities." *Scand. J. Work Environ. Health 21 (Suppl. 2)*:5-8 (1995).

[17] Vallyathan, V., Shi, X., Dalal, N.S., Irr, W., & Castranova, V.: "Generation of Free Radicals from Freshly Fractured Silica Dust: Potential Role in Acute Silica-Induced Lung Injury." *Am. Rev. Respir. Dis. 138*:1213-1219 (1988).

[18] Polichard, A., & Collet, A.: "Deposition of Siliceous Dust in the Lungs of the Inhabitants of the Saharan Regions." *Ind. Hyg. Occup. Med. 1945*:527-534.

[19] Vallyathan, V., Shi, X., Dalal, N.S., Irr, W., & Castranova, V.: "Generation of Free Radicals from Freshly Fractured Silica Dust: Potential Role in Acute Silica-Induced Lung Injury." *Am. Rev. Respir. Dis. 138*:1213-1219 (1988).

[20] Fubini, B., Bolis, V., Cavenago, A., & Volante, M.: "Physiochemical Properties of Crystalline Silica Dusts and their Possible Implication in Various Biological Responses." *Scand. J. Work Environ. Health 21 (Suppl. 2)*:9-14 (1995).

dictates that our risk assessment must rely on health effects reported in the occupational literature. Although freshly fractured silica content is more likely to occur in occupational settings, quantitative exposure measurements are presently unavailable from the research literature.

Particle size is an important consideration when estimating inhalation exposure. Generally, particles less than 10 μm in diameter enter the pulmonary system and those less than 5 μm enter deep alveolar spaces (air exchange spaces in the lung).[21,22] Small particle size and the assumption that all particulates below 10 μm are respirable represents a health conservative assumption and overestimates what would settle in the lung. In addition, these distribution assumptions may also be complicated by the general activity level, breathing rate, and ventilation style (whether a person breathes through the nose or mouth) and particulate shape.[23,24]

Seven biological responses are probable when dusts are taken into the lungs: 1) little to no reaction; 2) increased mucous secretion; 3) hypertrophy (enlargement) of mucous secreting glands; 4) macrophage ingestion; 5) chronic inflammatory reaction; 6) fibrosis; 7) malignant transformation of cells (cancer formation, cell metaplasia, or excessive cell division). Whether any of these reactions occurs depends on the individual, composition, quantity, and amount of all inhaled materials retained in the lungs. Respirable dusts deposited in the upper airways typically stimulate mucous secretion, trapping the particulate matter. These particulates are eventually removed from the lung. If extensive mucous secretions are not adequately removed, airway passages are narrowed. This condition promotes glandular enlargement and airflow resistance.

Dust particles inhaled deep into the lung stimulate macrophages in the alveoli. Macrophages ingest and remove particles from the lung. Ingestion of crystalline silica particulate may produce adverse effects or premature death for the macrophage. Products released by the damaged macrophage stimulate fibroblasts to produce excessive reticulin and collagen.[25] This process can lead to chronic inflammation and fibrosis. Malignant cell transformation is rare, but can follow in this sequence of biological changes in lung tissue. These events combine to increase the probability of malignancy. Respirable dust or other toxic agents are stimulants of this process.[26]

The focus of this report is silica and the material's capability to produce fibrotic disease. Other types of inorganic particulate (soot or carbon black) to which Gulf War veterans were exposed would fall in the low biological lung reaction category, meaning

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[21] Raabe, O.G.: "Deposition and Clearance of Inhaled Particles." *Occupational Lung Disease:*1-37 (1984).
[22] Rabovsky, J.: "Laboratory Studies on Silica Induced Toxicity and Relationship to Carcinogenicity." *J. Exposure Analysis and Environmental Epidemiology 7(3):*267-278 (1997).
[23] Parkes, W.R.: "Inhaled Particles and their Fate in the Lungs." In *Occupational Lung Disorders*, 2nd Ed., London/Boston: Butterworths, 1982. pp. 45-53.
[24] Raabe, O.G.: "Deposition and Clearance of Inhaled Particles." *Occupational Lung Disease:*1-37 (1984).
[25] Holian, A., Uthman, M.O., Golstova, T., Brown, S.D., & Hamilton, R.F.: "Asbestos and Silica-Induced Changes in Human Alveolar Macrophage Phenotype." *Environ Health Perspect. 105 (Suppl 5):*1139-1142 (1997).
[26] G.W. Wright: "The Pulmonary Effects of Inhaled Inorganic Dust" (Chapter 7). In *Patty's Industrial Hygiene and Toxicology*, 3rd Rev. Ed., Vol. 1 — General Principles. New York: John Wiley & Sons, 1978. pp. 175-176.

that extremely high exposure levels exceeding lung clearance capacity would need to be experienced well above current occupational exposure levels before an adverse biological response would be expected.

C. Sources of Respirable Particulate

The Kuwait Ministry of Public Health (KMPH) published a study of total particulates taken from four locations in the urban area of Kuwait distributed over 1089 days from 1980 to 1984.[27] The sampling sites represented a suburban industrial, a commercial city center, and two suburban residential localities. The overall mean total suspended particulate (TSP) levels were reported to be some of the highest in the published literature at that time. A total of 290 samples were collected and divided into two primary fractions, one greater than and one less than 7 microns. The respirable fraction (which they reported to be less than 7 microns) accounted for approximately 60% of the weight of the samples examined. Variability of the total weight of the samples was described as high and was found at all sites.

The U.S. Army reported on measurements of particulates in the air taken between May and December of 1991.[28] Their seven sampling locations included the area covered by the Kuwait Ministry of Public Health as well as locations within northwestern sections of Saudi Arabia. The mean, maximum, minimum, and 95% upper confidence limit (95% UCL) values for U.S. Army stations were similar to data presented in the KMPH study.[29] Analysis and conclusions of the present report rely heavily on the USAEHA sampling data. The USAEHA studies were conducted on the exposed population of concern and provided details on the composition of the particulate material inhaled for health risk determination.[30]

An overall environmental sampling and analysis effort was designed to assess the health risk from oil-well fires to Gulf War soldiers involved in Operation Desert Storm. Sand samples, ambient PM_{10} samples, and industrial hygiene samples were analyzed using a variety of analytical techniques. The specific purpose was to provide detailed particulate matter characterization. Continuous ambient air sampling of all personnel sites throughout the entire Gulf War Theater was impossible; therefore, sampling sites were selected based on high troop concentrations and long-term deployment positions. This resulted in sampling sites generally positioned downwind of oil fire locations.

This sampling strategy represents a conservative, worst-case scenario for Gulf War soldier particulate matter exposure. Representative data adequately characterize a best-fit statistical scenario. Each sample was used as part of a detailed human health risk

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[27] Kuwait Ministry of Public Health, Environment Protection Department: "Suspended Particulate Matter in the Urban Area in Kuwait." (1985). pp. 1-268.
[28] U.S. Army: "Final, Kuwait Oil Fire Health Risk Assessment." U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD, 1994. pp. 1-57.
[29] Kuwait Ministry of Public Health, Environment Protection Department: "Suspended Particulate Matter in the Urban Area in Kuwait." (1985). pp. 1-268.
[30] U.S. Army: "Final, Kuwait Oil Fire Health Risk Assessment." U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD, 1994. pp. 1-57.

assessment related to U.S. Military and Department of Defense civilian exposures to contaminants associated with the burning oil wells.[31]

Air sampling was completed by the U.S. Army at two stations in Kuwait: Camp Doha and the Ammunition Supply Depot in April and May of 1996. Particulate was sampled in the less than 10 micron range. These were analyzed for trace metals, volatile organic compounds (VOCs) related to hydrocarbons, and polycyclic aromatic hydrocarbons (PAHs). The concentration of particulate less than 10 microns was reported to be 80–120 $\mu g/m^3$ at Camp Doha and 50–100 $\mu g/m^3$ at the Ammunition Supply Depot. The concentrations of trace metals aluminum and iron in air samples taken during this study were in the 1–2 parts per billion (ppb) range, with the other metals in the parts per trillion (ppt) range or lower. There were detectable levels of some PAHs. The benzene, toluene, ethyl benzene, and xylene (BTEX) compounds totaled less than 8 $\mu g/m^3$. The levels of PM_{10} reported are generally below those reported by the U.S. Army in its 1996 study and the final report of environmental monitoring conducted during the 1990–1991 period of the Gulf War.[32]

1. Oil Fires and Other Combustion Products

Kuwaiti oil fires created unprecedented environmental pollution to which the population was exposed for a prolonged period. U.S. service members within the Gulf were exposed to these source pollutants of more than 600 burning oil-well fires. Majorities of burning wells were located in eastern Kuwait and south of Kuwait City. Smoke plumes rose and combined in a larger plume enveloping nearby troops. Pollutant plumes were composed of PAHs, benzene, various combustion residues, soot, and other aerosols. These combustion products added to the potential health concerns from naturally generated respirable particulate matter.[33]

Extensive air monitoring analysis was conducted for U.S. troop units exposed to these oil fire events. Risk assessment groups concluded that contributing oil fire and naturally generated respirable particulate pollutant levels were higher than anticipated and exceeded levels typical of industrial U.S. cities and EPA NAAQS. These pollutant levels, however, are not likely to cause long-term health effects.[34]

Consideration has also been given to identifying the possible health concerns from exposure to post-oil fire respirable particulate matter. Study results indicate a mixture of vapor phase pollutants and respirable particulates. The vapor phase pollutants, in combination with respirable particulates, may inhibit the defensive functions of lung alveolar phagocytes. This often leads to increased susceptibility to respiratory infections

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[31] U.S. Army: "Final, Kuwait Oil Fire Health Risk Assessment." U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD, 1994. pp. 1-57.
[32] U.S. Army: "Final, Kuwait Oil Fire Health Risk Assessment — Appendices A, B, G." U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD, 1994.
[33] U.S. Army: "Final, Kuwait Oil Fire Health Risk Assessment — Appendices A, I." U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD, 1994.
[34] U.S. Army: "Final, Kuwait Oil Fire Health Risk Assessment — Appendix I." U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD, 1994.

among the exposed population.[35] The inhalation of airborne products has been shown to alter respiratory function and affect lung cells, especially the alveolar phagocytes. These reactions are typical of diseases or conditions resulting from immune reactions in the lungs.[36] Chronic respiratory disease from short-term exposures would not be expected in the general military or civilian population. Only those who are highly sensitive (i.e., sufferers of severe chronic asthma) would possibly experience chronic disease impacts from short-term high exposures.[37]

2. *Sand Storms*

The U.S. Army Environmental Hygiene Agency conducted ambient air sampling in Kuwait and Saudi Arabia from May 1991 to December of 1991 for various pollutants emitted from the Kuwait oil-well fires. Approximately 4,000 ambient air samples were collected during this period from seven fixed sampling locations and two temporary sampling locations. Long-term sampling sites identified decreasing PM_{10} concentration levels from May through November of that year. Elevated PM_{10} levels are a result of sandstorms contributing to the overall measurable particulate concentrations. These fluctuations coincide with the Shamal wind season. A majority of the daily PM_{10} concentration levels observed at the sampling sites exceeded the PM_{10} 24-hour EPA NAAQS. The 24-hour PM_{10} standard is met when the 3-year average of the annual 99th percentile values at each monitoring site is less than or equal to 150 $\mu g/m^3$. These were exceeded for Riyadh, Jubayl, Ahmadi, Camp Thunderock, and the U.S. Embassy with a 24-hour mean PM_{10} range of 116 $\mu g/m^3$ to 301 $\mu g/m^3$.[38]

3. *Daily Operations*

U.S. troops were exposed during daily activities to petroleum fuels, their combustion products, and other operational-related air pollutants. Operating vehicles and machinery used in the Gulf conflict involved exposure to petroleum-based substances and increased levels of combustion particulates. Petroleum fuels were also used for burning wastes and trash, dust suppression, and for fueling stoves and tent heaters. Even though none of the uses was unique to the Gulf War, it probably led to increased petroleum vapor and combustion products, including particulates. Although dust suppression operations may have led to higher petroleum vapor exposures (use of crude oil, waste oil, and diesel oil), the level of respirable dust would be reduced. In desert military operations, high respirable dust levels during troop and vehicle movement is often a major problem. Vehicle exhaust exposures, such as diesel exhaust, are common during heavy equipment movement. In the absence of USAEHA dust monitoring data for some of these situations, it seems reasonable to assume that dust exposures would have been

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[35] Ezeamuzie, C.I., Beg, M.U., & Al-Ajmi, D.: "Responses of Alveolar Macrophages to Post-Gulf-War Airborne Dust from Kuwait," *Environment International*, Vol. 24(1/2), pp. 213-220 (1998). [Elsevier Science Ltd.]

[36] Milanowski, J.: "Effect of Inhalation of Organic Dust-Derived Microbial Agents on the Pulmonary Phagocytic Oxidative Metabolism of Guinea Pigs." *J. Toxicol. Environ. Health 53*:5-18 (1998).

[37] Milanowski, J.: "Effect of Inhalation of Organic Dust-Derived Microbial Agents on the Pulmonary Phagocytic Oxidative Metabolism of Guinea Pigs." *J. Toxicol. Environ. Health 53*:5-18 (1998).

[38] U.S. Army: "Final, Kuwait Oil Fire Health Risk Assessment — Appendices B, G." U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD, 1994.

high for short periods and might have caused short-term irritations, but not long-term problems. A research suggestion for the future is to conduct short-term industrial hygiene particulate exposure monitoring in some of these potentially dusty work situations and evaluate the need for corrective action. In addition, acute animal studies of an inhaled mixture of silica, petroleum hydrocarbons, and soot would be helpful.

V. ENVIRONMENTAL MEASUREMENTS

A. U.S. Standards and Guidelines

The U.S. Permissible Exposure Limit (PEL) for occupational exposure to silica is 0.1 mg/m^3 (based on a rate of 40 hours per week for 70 years). It was established for the most common type of quartz: alpha-quartz. Less abundant and more toxic species include the crystalline polymorphs: cristabolite, tridymite, and tripoli. Currently, OSHA and the Mine Safety and Health Administration (MSHA) use the American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Values (TLVs®) as their PELs. These agencies also consider the TLV to be a valid limit for mixtures containing low percentages of silica. ACGIH has set TLVs for silica exposure depending on the species of silica in the workplace (see Table 1).[39]

Table 1. Threshold Limit Values (TLVs®) for selected silica species.

Silica Species	Threshold Limit Value
Cristabolite	**0.05 mg/m^3**
Quartz	**0.1 mg/m^3**
Tridymite	**0.05 mg/m^3**

There is considerable support that silica exposure guidelines of either 0.1 mg/m^3 or 0.05 mg/m^3 are adequate for short-term occupational exposure (10 years or less).[40] The OSHA PEL and the MSHA recommended limit are based on a formula (10% of quartz + 2). Assuming that the crystalline silica is quartz — the form of importance to Gulf War veterans — and its percentage is 100%, the dust will result in a concentration of 0.1 mg/m^3 for cristabolite and tridymite, which are considered the more pathogenic forms of silica. Occupational studies have identified negligible risk of silicosis from exposures at or below 1 mg/m^3 × year (see Figure 3).[41]

New research has examined the potential health concerns resulting from exposure to both the 0.1 mg/m^3 (ACGIH TLV, MSHA limit, OSHA PEL), and the 0.05 mg/m^3 (NIOSH REL) respirable silica levels. ACGIH published a 0.05 mg/m^3 TLV for the first quarter of 2000 and the A2 "suspected human carcinogen" designation for silica was

[39] National Institute for Occupational Safety and Health: *NIOSH Pocket Guide to Chemical Hazards.* Cincinnati, OH: NIOSH, 1990. pp. 194-197.

[40] Banks, D.E., Morring, K.L., Boehlecke, B.A., Althouse, R.B., & Merchant, J.A.: "Silicosis in Silica Flour Workers." *Am. Rev. Respir. Dis. 124:*445-450 (1981).

[41] Gift, J.S., & Faust, R.A.: "Noncancer Inhalation Toxicology of Crystalline Silica: Exposure-Response Assessment," *J. Expos. Analysis Environ. Epidemiol. 7(3):*345-358 (1997).

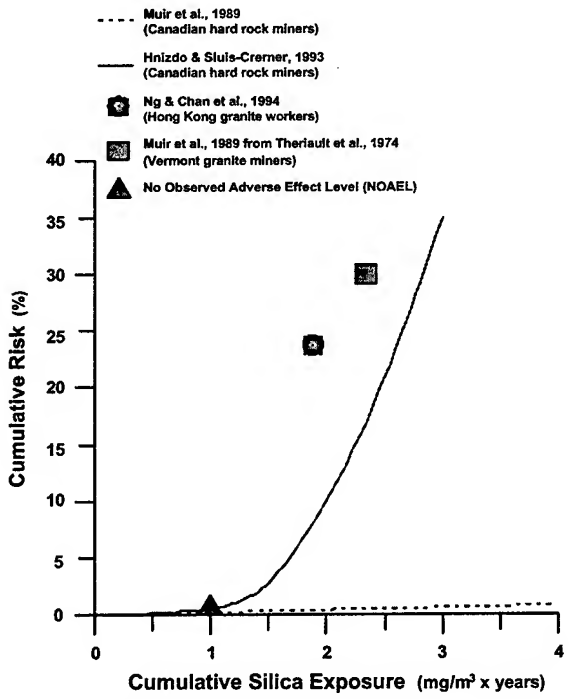

Figure 3. Health effect risks are negligible at or below 1 mg/m^3 × year silica exposure levels. Likelihood of silicosis is estimated from exposure levels and reported as cumulative risk. Representative occupational studies and the no observed adverse effect level (NOAEL) are plotted against cumulative risk estimates (South African gold miners, Hnizdo and Sluis-Cremer; 1991; Canadian hardrock miners, Muir et al., 1989; Hong Kong granite workers, Ng and Chan, 1994; Vermont granite miners, Theriault et al., 1974; Muir et al., 1989).

included.[42] Questions remain about the adequacy of either of these levels to prevent long-term pulmonary health effects in workers. Current research indicates that neither of these levels may be adequate as a NOAEL for long-term occupational exposure. According to recent studies, health effects may occur at cumulative respirable silica exposures below 0.05 mg/m^3 for a working lifetime of 45 years.[43]

The EPA accepted methods for dose–response and risk assessment are used to convert occupational standards to ambient conditions. It is determined that an ambient silica exposure in PM_{10} between 3 µg/m^3 and 8 µg/m^3 (U.S. range for a continual TWA) would be the equivalent of 8.4 µg/m^3 and 22.4 µg/m^3 for an occupational level.

Such exposure levels would result in 0.6–1.6 mg/m^3 × year of cumulative silica exposure for a 70-year lifetime and a 0.03%–2.4% upper bound risk of developing silicosis. This equates to a risk of 0.3% at 1 mg/m^3 × year cumulative exposure (continually breathing 0.005 mg/m^3 of silica in PM_{10} for 70 years).[44]

The formula EPA used to convert ambient exposure to an occupational exposure was: continuous exposure = occupational exposure × (5 days/7 days) × (10 m^3/20 m^3 of air breathed/day). Because the continuous ambient exposure was known and the occupational equivalent exposure needed to be calculated so that EPA could evaluate health risks to the general public from the occupational health database on silica, the formula EPA used was: occupational (equivalent) exposure = continuous exposure × (7-day week/5-day week) × (20 m^3/10 m^3 of air breathed/day), or a factor of 2.8 multiplied by the ambient continuous exposure. In the EPA study, cumulative exposure was defined

[42] American Conference of Governmental Industrial Hygienists: *ACGIH Today — Annual Report,* Vol. 7, Issues 7 and 8 (December 1999).

[43] Gift, J.S., & Faust, R.A.: "Noncancer Inhalation Toxicology of Crystalline Silica: Exposure-Response Assessment," *J. Expos. Analysis Environ. Epidemiol. 7(3):*345-358 (1997).

[44] U.S. EPA/Office of Research and Development: "Ambient Levels and Noncancer Health Effects of Inhaled Crystalline and Amorphous Silica: Health Issue Assessment" (EPA/600/R–95/115). November 1996. pp. 7-1–7-6.

as occupational equivalent exposure × 70 years.[45] In our analysis of USAEHA exposure data, we use the same approach as EPA with an adjustment upward for a slightly higher breathing rate of 24 m^3/day and a necessary time-in-field compensation factor for the less-than-1-year average duration for the Gulf War veterans.

EPA further calculated a 70-year lifetime ambient cumulative exposure to be in the range of 0.6–1.6 mg/m^3 × year, resulting in a benchmark dose analysis. EPA subsequently calculated an upper bound risk of developing silicosis between 0.03% and 2.4%, with a 0.3% risk resulting from an average of 5 µg/m^3 respirable silica breathed continually over a 70-year lifetime. EPA calculated upper bound risk levels (cumulative risk) using a log-logistic model to estimate the lower bounds of cumulative exposure and benchmark doses (BMD) that may cause health effects.[46] One of the fundamental scientific studies reviewed indicated that an ambient NOAEL from respirable silica dust inhaled over an average lifetime (70 years) would be less than or equal to 1 mg/m^3 × year, or about 3,000 mg for a lifetime.[47]

B. Air Monitoring Programs and Results

Examination of the USAEHA 1991 and the USAEHA 1994 reports revealed that industrial hygiene and PM_{10} air samples taken at the time of the Gulf War contained low amounts of respirable silica. A low level of respirable silica in an environment of sand seems counterintuitive to what one would believe, but the literature review supports this conclusion.

Researchers determined that the Northern Saharan desert dust particle spectrum between 0.1 micron and 20 micron desert dust was composed of clays. They also noted that silica was nearly absent from the finer respirable dust particles. The clay-rich, silica-poor composition of respirable dust was consistent regardless of the weather conditions.[48] Littmann's study of respirable dust in the Israel Negev desert noted that deposited dust did not differ in composition from background dust, and those smaller particulate sizes had lower silica content. A regional dust sample with a grain size of 91% in the PM_{10} range contained only 9% quartz, compared with a sample that contained only 5.9% in the PM_{10} range yet contained 83.5% quartz. Therefore, the relationship of decreasing silica content with decreasing particle size supports the use of values less than 10% as reasonable estimates of silica content in respirable dust or inhaled material below 10 microns in diameter.[49] USAEHA studies, which were representative of sand particulates

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[45] U.S. EPA/Office of Research and Development: "Ambient Levels and Noncancer Health Effects of Inhaled Crystalline and Amorphous Silica: Health Issue Assessment" (EPA/600/R–95/115). November 1996. pp. 7-1–7-6.

[46] U.S. EPA/Office of Research and Development: "Ambient Levels and Noncancer Health Effects of Inhaled Crystalline and Amorphous Silica: Health Issue Assessment" (EPA/600/R–95/115). November 1996. pp. 7-1–7-6.

[47] Gift, J.S., & Faust, R.A.: "Noncancer Inhalation Toxicology of Crystalline Silica: Exposure-Response Assessment," *J. Expos. Analysis Environ. Epidemiol. 7(3):*345-358 (1997).

[48] Gomes, L., & Bergametti, G.: "Submicron Desert Dusts: A Sandblasting Process." *J. Geophysical Research 95(D9)13:*927-935 (August 1990).

[49] Littmann T.: "Atmospheric Input of Dust and Nitrogen into the Nizzana Sand Dune Ecosystem, North-western Negev, Israel." *J. Arid Environments 36:*433-457 (1997).

in the Gulf War Theater, confirmed a similar composition to other desert areas reviewed in the scientific literature. Bulk samples contained an average of 4.3% and a maximum of 6.5% alpha-quartz in the silicon-rich portion of respirable particulate matter (less than 10 microns).[50] Silicon-rich here refer only to silicon-oxygen compounds consisting of amorphous and crystalline silica; silicates and clays were separately identified by analytical means from the silica portion. The amount of silica may be overestimated, but it would not exceed the levels estimated in the report.[51] These values were used to estimate respirable silica exposures in this report.

VI. HEALTH EFFECTS

This health effect literature review focused on respirable silica and, to a lesser extent, on dust or TSP. Exposure documentation showed these areas to be of the greatest potential health concern to Gulf War soldiers. Primarily, the USAEHA findings document levels greater than the EPA recommended TSP and respirable dust exposure levels. The study by the Kuwait Public Health Ministry concluded with evidence that the Kuwaiti people were exposed to large amounts of respirable dust and that the high incidence of respiratory difficulties such as asthma were related to the high dust levels.[52]

A literature review of the health effects of components besides silica that were previously identified for inhaled particulate materials for Gulf War soldiers has consistent findings. Multiple international agency and U.S. studies identified oil fire pollutant levels and exposures as not-likely sources for long-term health effects.[53] Participating risk assessment groups concluded that levels of nitrogen oxides, carbon monoxide, sulfur dioxide, hydrogen sulfide, PAHs, benzene, and other surprisingly typical airborne pollutants in most cases did not exceed those found in urban air in a typical U.S. industrial city.[54]

Samples were collected during at least one instance when the smoke plume had touched down, providing a worst-case exposure. This incident produced detectable airborne contaminants, although the levels were low when compared with current U.S. occupational standards for these contaminants.[55]

Common occupational operations such as sandblasting, tunneling, drilling, and silica milling produce silica particulates in high concentrations. These industrial exposures produce crystalline quartz particles that are cleaved or sheared and are more

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[50] U.S. Army: "Final, Kuwait Oil Fire Health Risk Assessment — Appendix G." U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD, 1994.
[51] Private conversation with Gary Cassucio, analyst with R.J. Lee, November 2, 1998.
[52] Kuwait Ministry of Public Health. Environment Protection Department: "Suspended Particulate Matter in the Urban Area in Kuwait," 1985. pp. 1-268.
[53] U.S. Army: "Final, Kuwait Oil Fire Health Risk Assessment — Appendices G, I." U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD, 1994.
[54] U.S. Army: "Final, Kuwait Oil Fire Health Risk Assessment — Appendices A, B, G, I." U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD, 1994.
[55] U.S. Army: "Final, Kuwait Oil Fire Health Risk Assessment — Appendices A, B, G, I." U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD, 1994.

biologically reactive than aged silica.[56] Exposure of soldiers in the Gulf to natural silica is believed to be less dangerous than industrial operations where the majority of health effects data exist. Most occupational exposure arises from man-made operations. These operations create greater amounts of freshly fractured silica when compared to naturally occurring conditions. Gulf War soldier silica exposures were primarily of natural origin. It is unlikely to have a high, freshly fractured content. At this time, however, we cannot be certain of how much freshly fractured silica was a part of particulate exposure. Thus, the biological reactivity of Persian Gulf sand cannot be quantified because little is known about its freshly fractionated silica portion in the natural desert environment. Based on the health conservative purposes of this analysis, the authors considered all silica — natural or man-made — equal in its ability to generate health effects. All silica was considered to be biologically reactive.

A. Troop Medical/Hospital Studies

A few studies have been conducted examining the activities and experiences of physicians, nurses, and other medical personnel in treating soldiers during the Gulf War. The compilation of clinical data is generally difficult because of the frequent transfers of patients and a lack of coordination of medical records. A common ailment that occurred among Gulf War soldiers was "desert dust asthma."[57] This was described as a protracted asthmatic bronchitis. The symptoms were dyspnea (shortness of breath), cough, rales, and wheezing. Spirometry showed mild to moderate airway obstruction, which was not improved by bronchodialator therapy, but was improved with steroid treatment. Fine respirable desert dust and dryness contributed to this condition. Examination of the Desert Shield/Desert Storm Medical Evacuation DataBase (August 1990–August 1992) showed a total of 8,046 medically related evacuations of individuals, and 461 (5.7%) of those evacuations were of a respiratory-related diagnosis (e.g., 212 individuals with unspecified asthma). Some of these evacuations may have been related to pre-existing respiratory ailments such as chronic asthma. For example, the incidence of allergic reactions in the general U.S. population has been estimated to range from approximately 3% to 10% of the population, but it would vary from one section of the country to another.[58] Soldiers with these medical conditions, which may have existed before the Gulf War, would be more susceptible to the effects of desert dust, dryness, and airborne contaminants than military personnel in general.

B. Development of Silicosis

By definition, silicosis (or "grinders disease") is an occupationally based pulmonary disease. Silicosis, as with many acute and chronic pulmonary diseases, is directly related to harmful substance inhalation. Workplace environments bring individuals into contact with harmful substances promoting pulmonary disorders,

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[56] Vallyathan, V., Castronova, V., Pack, D., Leonard, S., Shumaker, J., Hubbs, A.F., Shoemaker, D.A., Ramsey, D.M., Pretty, J.R., McLaurin, J.L., Kahn, A., & Teass, A.: "Freshly Fractured Quartz Inhalation Leads to Enhanced Lung Injury and Inflammation." *Am. J. Respir. Crit. Care Med. 152:*1003-1009 (1995).

[57] Pohlmann, G.P.: "War and Medicine in the Desert, A Report of the 13th Evacuation Hospital in Saudi Arabia." *Wisconsin Medical Journal:*511-515 (September 1991).

[58] NRC: "Markers in Immunotoxicology," 1992. p. 37.

including: 1) pneumoconiosis; 2) hypersensitivity pneumonitis; 3) obstructive airway disorders; 4) toxic lung injury; 5) lung cancer; 6) pleural diseases; and 7) miscellaneous disorders. Pneumoconiosis is a chronic fibrotic lung disease promoted by inhalation of respirable coal dust and various inert, inorganic, or silicate dusts (see Table 2).[59]

Table 2. Selected Pneumoconiosis

Disease	Agent	Occupational Sources
Siderosis	Metallic iron or iron oxide	Mining, welding, foundry work
Stannosis	Tin, tin oxide	Mining, tin work, smelting
Baritosis	Barium salts	Glass and insecticide manufacturing
Pneumoconiosis	Coal dust	Coal mining
Silicosis	Free silica (silicon dioxide)	Rock mining, quarrying, stone cutting, tunneling, sandblasting, pottery, diatomaceous earth
Asbestosis	Asbestos	Mining, insulation, construction, shipbuilding
Talcosis	Magnesium silicate	Mining, insulation, shipbuilding
Kaolin pnuemoconiosis	Sand, mica, aluminum silicate	China clay mining, pottery, cement work
Shaver's disease	Aluminum powder	Manufacture of corundum

Silicosis occurs when extensive or prolonged inhalation of free silica (silicon dioxide) particles in the respirable range (0.3–10 microns) causes the formation of small rounded opacities (silicotic nodules) throughout the lung. Calcification of the hilar lymph node periphery ("eggshell" calcification) is an unusual finding that strongly suggests silicosis. Simple silicosis is usually asymptomatic and has no effect on routine pulmonary function tests. Complicated silicosis produces large conglomerate densities in the upper lung. These are accompanied by dyspnea and obstructive/restrictive pulmonary dysfunction.

C. Mechanism of Toxicity

Acute silicosis is characterized by increases in pulmonary phospholipid, inflammatory cells (neutrophils and lymphocytes), damage at the blood-alveolar air barrier level (air exchange barrier), and activation of oxidant production by pulmonary macrophages. Generation of reactive oxygen species in the lung overwhelm the antioxidant defense system to cause endothelial cell injury and edema.

Compared to aged silica, freshly ground silica exhibits greater cytotoxic effects on cellular membranes.[60] Precise mechanisms whereby peroxidation causes cytotoxicity and necrosis are not well defined or accepted. The three possible mechanisms for this damage are:

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[59] Tierney, L.M., McPhee, S.J., & Papadakis, M.A.: "Lung". In *Current Medical Diagnosis and Treatment*, 34th Ed. Norwalk, CT: Appleton and Lange, 1995; pp. 203-280.

[60] Vallyathan, V., Shi, X., Dalal, N.S., Irr, W., & Castranova, V.: "Generation of Free Radicals from Freshly Fractured Silica Dust: Potential Role in Acute Silica-Induced Lung Injury." *Am. Rev. Respir. Dis. 138*:1213-1219 (1988).

- Breakdown products of peroxidized lipids (such as 4-hydroxynonenal) are cytolytic and destroy the membrane.
- Radical products of peroxidation may covalently bind to essential cellular components.
- Peroxidation of the plasma membrane may cause sufficient change in its physical properties that certain functions, namely calcium homeostasis, are lost.

D. Dermal Toxicants

Dermal exposures to silica dust are also considered in this report. Silica dusts are associated with specific types of dermatitis or skin inflammation. A dry, dusty environment can promote minor skin reactions. These irritations, however, are not expected to initiate long-term chronic skin disorders. Recent studies associate connective tissue diseases, especially scleroderma or dermatosclerosis, with prolonged exposure to silica. Scleroderma is characterized by formation of thickened fibrous tissue and adhesion to the underlying tissues. These conditions commonly affect hand and facial tissue. Long-term exposures to silica dusts can produce progressive systemic scleroderma. It should be noted that retained and inhaled particles of crystalline silica can be transported to other organs through the lymphatic system, causing cellular immune responses believed to be involved as a risk factor for connective tissue diseases.[61] This is commonly diagnosed when the symptoms are widespread to include the entire body.[62]

Despite a known correlation between this systemic skin disorder and silica dust exposure, time and silica concentration exposures are critical factors in the development of this disease. Long-term dermal exposure times are required before scleroderma symptoms occur. Various studies identify required exposure times between 3 and 34 years.[63] Even though there are differences of opinion about true cause-and-effect relationships between crystalline silica exposure and extrapulmonary effects such as scleroderma, persuasive evidence is present relating scleroderma to an increased risk of silicosis in occupational settings. No increased evidence of an extrapulmonary condition has been presented at low crystalline silica exposure levels.[64] Gulf War soldiers were not exposed to silica dust content greater than three-year duration or at levels approaching occupational settings where silicosis is present.[65] Therefore, health risks resulting from dermal exposure are negligible.

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[61] "Adverse Effects of Crystalline Silica Exposure" (Official Statement of the American Thoracic Society). Published in the *American Journal of Respiratory and Critical Care Medicine,* Vol. 155 (June 1996). p. 764.

[62] Cowie, R.L.: "Silica-Dust-Exposed Mine Workers with Scleroderma (Systemic Sclerosis)." *Chest 92(2)*:260-262 (August 1987).

[63] Anandan, S., Othman, M., Cheong, I., & Chin, G.L.: "Scleroderma Secondary to Silica Exposure — A Case Report." *Singapore Med. J. 36(5)*:559-561 (October 1995).

[64] "Adverse Effects of Crystalline Silica Exposure" (Official Statement of the American Thoracic Society). Published in the *American Journal of Respiratory and Critical Care Medicine,* Vol. 155 (June 1996). p. 764.

[65] Haustein, U.F., Ziegler, V., Herrman, K., Mehlhorn, J., & Schmidt, C.: "Silica-Induced Scleroderma." *J. Am. Acad. Dermatol. 22(3)*:444-448 (March 1990).

E. Potential Hazards

Studies in laboratory animals and humans indicate that pulmonary inflammatory response, fibrogenic and silicotic lung lesions are the primary pathological result from the inhalation of crystalline silica. Lung cancer is a probable secondary pathological concern to the formation of fibrotic lesions. Epidemiological studies show small increased relative risks of lung cancer in workers exposed over many years to crystalline silica.[66]

Silica exposure limits of 0.05 mg/m^3 are adequate to protect workers for short-term occupational exposures of 10 years or less. There is considerable support in the scientific literature for this conclusion. If the limit of 0.05 mg/m^3 is used, the cumulative silica NOAEL of 1 $mg/m^3 \times$ year for occupational silica exposure can be calculated. Occupational studies have identified negligible risk of silicosis from exposures at or below 1 $mg/m^3 \times$ year.[67]

1. Soot

Respired carbon-based particulate matter, or soot, is an important area to examine for potential health risks to Gulf War veterans. Soot is a combination of particles impregnated with tar and formed by the incomplete combustion of organic materials. Approximately 600 Kuwaiti oil wells were set ablaze toward the end of the Gulf War, releasing an estimated 20,000 tons of soot.[68] USAEHA air particulate measurements estimated that 23% of the particulate matter sampled during the Gulf War was soot from Kuwaiti oil fires.[69] Soot particulate concentrations were less than 10% of air samples taken from Saudi Arabia.[70]

Calculations based on smoke from Kuwaiti oil fires in May and June of 1991 indicate that combustion efficiency was about 96% in producing carbon dioxide. Smoke particulate matter accounted for 2% of the fuel burned, of which 0.4% was soot — or 20% of the total fuel burned by weight. Actual measurements of soot concentration were 0.45% of the fuel burned, or 0.53% of the total carbon emitted.[71] VOCs and polycyclic aromatic hydrocarbons (PAHs) were lower in Gulf War regions than levels found in most U.S. urban areas and below recommended exposure levels.[72] Combustion efficiency combined with low VOC and PAH levels are useful in characterizing the soot particulate

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[66] Klein, A.K., & Christopher, J.P.: "Evaluation of Crystalline Silica as a Threshold Carcinogen." *Scand. J. Work Environ. Health 21 (Suppl. 2)*:95-98 (1995).
[67] Gift, J.S., & Faust, R.A.: "Noncancer Inhalation Toxicology of Crystalline Silica: Exposure-Response Assessment." *J. Expos. Analysis Environ. Epidemiol. 7(3)*:345-358 (1997).
[68] Husain, T.: *Kuwaiti Oil Fires: Regional Environmental Perspectives.* Oxford, U.K.: Pergamon Press Ltd., 1995.
[69] Spektor, D.M.: "A Review of the Scientific Literature as it Pertains to Gulf War Illness — Oil Well Fires," *RAND*, Vol. 6 (1998).
[70] Spektor, D.M.: "A Review of the Scientific Literature as it Pertains to Gulf War Illness — Oil Well Fires," *RAND*, Vol. 6 (1998).
[71] Spektor, D.M.: "A Review of the Scientific Literature as it Pertains to Gulf War Illness — Oil Well Fires," *RAND*, Vol. 6 (1998).
[72] Spektor, D.M.: "A Review of the Scientific Literature as it Pertains to Gulf War Illness — Oil Well Fires," *RAND*, Vol. 6 (1998).

content in the Gulf. USAEHA air sampling and analysis provide comparisons with health effects of various soot materials identified in the scientific literature.

Similarities in composition, production, and reviewed documentation indicate a scientifically reasonable comparison between Kuwaiti oil-fire soot and carbon black. The soot component of particulate matter has characteristics similar to — and may reasonably be compared with — carbon black. This assessment recognizes that carbon black is formed in a controlled industrial manufacturing process. Consideration is also given to differences between uncontrolled combustion products normally characterized as soot and manufactured carbon black. However, similarities between carbon black and substances present in Gulf War soot are significant enough to warrant toxicological comparison between the health effects from exposure to these two materials.

Analytical data, for example, identify both carbon black and Gulf War soot with low VOC, low PAH, and high carbon content. USAEHA noted that "soot in the plumes is elemental carbon in the form of small carbon chain agglomerates"[73] — an important fact for comparison of Gulf War soot to carbon black and other combustion material.

Carbon black is a fine respirable material with oily lubricant-type properties. Considerable human health effects data and scientifically sound occupational exposure standards are established for carbon black. Greater than 85% of carbon black is elemental carbon obtained by hydrocarbon decomposition or combustion. Carbon black produced in the United States is used in the rubber, ink, paint, and paper industries. If workplace carbon black PAH and VOC concentrations exceed 0.1% by weight, carbon black is handled as a suspect carcinogen.[74]

Three properties were examined in the literature for comparing the toxicological significance of soot produced in the Gulf War with commercial carbon black:

1) *Low levels of polynuclear aromatic hydrocarbons were present in Gulf War soot particulate matter, well below 0.1%.* Analysis of smoke from Kuwaiti oil fields showed total PAH levels of 16 $\mu g/m^3$ and benzo(a)pyrene (BaP) levels assessed at 19 µg/g of smoke. The PAH concentration in dust from air conditioners collected during the Gulf War in residential areas was no higher than 16.7 µg/g and then dropped to an ambient level of 2.4 µg/g a year later. In northern Kuwait, the highest PAH sample collected was 11.1 µg/g.[75] Smoke samples during the war would be 0.0016% BaP and the same concentration ranges for PAHs in the air-conditioning dust. The mass/mass concentration ratio of PAHs from these external studies is approximately 100× below the level where one would consider carbon

[73] USAEHA: "Final Report–Kuwaiti Oil Fire Health Risk Assessment (No. 39–26–1192–91, 5 May–3 December 1991), Appendix G — Sand and Ambient Air Sample Analysis, Vol. 1. p. G-14.
[74] National Institute for Occupational Safety and Health: "Criteria for a Recommended Standard, Occupational Exposure to Carbon Black" (NIOSH Pub. No. 78–204). Cincinnati, OH: NIOSH, 1978. p. 107.
[75] Saeed, T., et al.: "Comparison of the Levels of PAHs Present in the Dust Collected from Different Residential Areas of Kuwait, 1991–1996," *Environment International 24:*197; 1998. [Elsevier Science Ltd.]

black soot a suspect carcinogen and does not approach 0.1%. USAEHA studies indicated that the majority of PAH hydrocarbons were below detectable limits in samples taken in Saudi Arabia and Kuwait — only naphthalene was found above the detection limit in three samples.[76] Data indicating a low PAH content in air and in the particulate material is a significant toxicological criterion for comparing a carbonaceous particulate from any combustion scenario to a carbon black.

2) *Carbon particles produced in Gulf War soot and commercially produced carbon blacks show similarity in particle shape and size.* Carbon black is produced by a number of commercial processes, one of which is thermal decomposition (combustion) of aromatic oil and coal tar distillates to produce carbon particles in the 0.01 μm–0.5 μm range, with thermal decomposition producing generally larger soot particles in the 0.12–0.5 μm range.[77] USAEHA found that Gulf War soot was a mixture of small carbon chain agglomerates of 0.1 μm and larger carbon chains composed of spherical particles 0.25 μm or greater.[78] Since Gulf War soot was produced by petroleum thermal decomposition, carbon chain agglomeration with spherical carbon particles similar to commercial carbon blacks would be expected.

3) *Evidence of low bioavailability of organic materials (VOCs) was present in the Gulf War soot.* Commercial carbon blacks range in purity from 85% to 99% carbon.[79] The soot produced in Gulf War fires was not that pure. Chemical analysis of soot by USAEHA indicated organic carbon in addition to elemental carbon particles averaged 45% of the total carbon present in PM_{10} samples. This is above the organic range for carbon black. Some samples were very high in elemental carbon and low in organic carbon. One example was an Ahmadi hospital where a soot sample was 81% elemental carbon, 27% organic, and 1% carbonate. Most PM_{10} samples, however, contained a higher organic carbon to elemental carbon concentration ratio.[80] Although organic carbon was present in particulate air samples, the soot particulate itself was mostly elemental carbon.[81] This analysis is important because it indicates that though some Gulf War soot particles might not be as chemically pure as carbon black, the organic materials present were tightly bound and chemically unextractable. Since

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[76] USAEHA: "Interim Report–Kuwaiti Oil Fire Health Risk Assessment (No. 39–26–1192–91, 5 May–3 December 1991), Appendix D — Industrial Hygiene Air Sampling, Vol. 1, p. D-8(2).

[77] Brockmann, M.;Fisher, M.; & Mueller, K.-M.: "Exposure to Carbon Black: A Cancer Risk?" *Int. Arch. Occup. Environ. Health 71:* 85-99 (1998).

[78] USAEHA: "Interim Report–Kuwaiti Oil Fire Health Risk Assessment (No. 39–26–1192–91, 5 May–3 December 1991), Appendix G — Sand and Ambient Air Sample Analysis, Vol. 1, p. G-30.

[79] National Institute for Occupational Safety and Health: "Criteria for a Recommended Standard ... Occupational Exposure to Carbon Black" (NIOSH Pub. No. 78–204). Cincinnati, OH: NIOSH, 1978. p. 107.

[80] USAEHA: "Final Report–Kuwaiti Oil Fire Health Risk Assessment (No. 39–26–1192–91, 5 May–3 December 1991), Appendix G — Sand and Ambient Air Sample Analysis, Vol. 1. Table G-2-18.

[81] USAEHA: "Final Report–Kuwaiti Oil Fire Health Risk Assessment (No. 39–26–1192–91, 5 May–3 December 1991), Appendix G — Sand and Ambient Air Sample Analysis, Vol. 1. p. G-14.

some organic materials should have been detected at significant particulate loading with CS_2 extraction, their absence indicates that Gulf War soot had significant adsorbent properties for organics, or possibly the organic materials were of sufficiently high molecular weight that they were unextractable by carbon disulfide. Either explanation supports an assumption that organics were likely not bioavailable, lending evidence to support that the toxicological behavior of Gulf War soot particulate material was similar to elemental carbon, or an inert carbon black.

Reduced pulmonary function and respirable irritation are the main occupational and toxicological health effects from carbon black. These symptoms are expressed after exposure to large concentrations over long periods of time. A 1978 NIOSH criteria document investigated toxicological and epidemiological evidence to revise the carbon black standard. This document suggested carbon black might cause adverse pulmonary, cardiovascular, and dermal effects at higher concentrations, but it concluded that the present limit of 3.5 mg/m^3 should be maintained. Free-radical activity and inflammatory effects of PM_{10} and carbon black have been examined using intratracheal instillation in rats. Results indicate that PM_{10} produces a greater inflammatory response than fine carbon black.[82]

The current report considered effects of soot or carbon black as a separate and inert dust exposure. Because a scientific basis for any synergistic effects within the lung is lacking, the possible additive effects of silica and other respirable materials were considered. This reasoning is based on differences in pulmonary effects among different types of particulates. Respirable carbon black, unlike silica, does not promote pulmonary fibrosis. Carbon black is a carbon-based dust. When inhaled by experimental animals, these materials produce little or no collagen and, in all respects, can be classified as an inert dust.[83] In contrast, inhaled silica promotes pulmonary fibrosis, a common secondary result of chronic pulmonary inflammation and collagen formation. Excessive collagen formation accompanies prolonged or chronic inflammation in most organs of the body. This is associated with scar tissue and reduced pulmonary function.

The OSHA PEL for carbon black is 3.5 mg/m^3. The NIOSH REL and the ACGIH TLV® are the same. In 1978, a review by NIOSH determined that the data at that time did not support a reduction in the existing federal limit of 3.5 mg/m^3 and proposed that the limit be maintained.[84] ACGIH has not placed carbon black on its notice of intended TLV changes for 1999. The 3.5 mg/m^3 occupational exposure limit remains scientifically justified based on current toxicological research.

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[82] Li, X.Y., Gilmour, P.S., Donaldson, K., & MacNee, W.: "Free Radical Activity and Pro-Inflammatory Effects of Particulate Air Pollution (PMl0) In Vivo and In Vitro." *Thorax 51(12)*:1216-1222 (1996).

[83] Clayton, D.G., & Clayton, F.E.: *Patty's Industrial Hygiene and Toxicology,* 3rd Rev. Ed., Vol. 1. New York: John Wiley & Sons, 1978.

[84] National Institute for Occupational Safety and Health: "Criteria for a Recommended Standard ... Occupational Exposure to Carbon Black" (NIOSH Pub. No. 78–204). Cincinnati, OH: NIOSH, 1978. p. 107.

As with silica, the largest and most scientifically sound database for human health effects from carbon black exposure is the occupational health literature. Persons do not live or spend entire lifetimes with continual exposure in an extremely dusty, smoke-filled or high soot environment. Therefore, there is no ambient carbon black exposure level for a 24-hour continual exposure. In this report, it was estimated from the occupational health effects database. Using 3.5 mg/m^3 as a conservative health limit and as an occupational NOAEL, a 45-year occupational lifetime would produce a cumulative worker exposure of 157 mg/m^3 × year. Calculating an estimated 24-hour/7-day/week continuous exposure NOAEL by applying the EPA conversion formula as described in Section V of this report (Environmental Measurements), it is estimated that a continuous ambient (environmental) carbon black exposure level would be 1.25 mg/m^3 (3.5 mg/m^3 × [5 days/7 days × 10 m^3 breathed per day/20 m^3 breathed per day] = 1.25 mg/m^3). Over a 70-year lifetime, this would amount to an acceptable cumulative exposure of 87.5 mg/m^3 × year of ambient carbon black. If the work year were reduced from 52 weeks to 45 weeks, this number would be reduced slightly (14%) to 75.7 mg/m^3 × year of ambient carbon black. This would be as total particulate, not just respirable material. Given the fine nature of carbon black, however, it would be reasonable to assume that most — if not all of it — is respirable. This is a relatively large allowable cumulative exposure to an inhaled particulate, even though the particulate is considered a biologically inert dust. Environmental measurements during Kuwaiti oil fires revealed mean concentrations of soot from smoke plumes between 0.036 mg/m^3 and 0.308 mg/m^3.[85] This is quite low when compared with adjusted ambient environmental exposure limits of 1.25 mg/m^3 for carbon black, as calculated above. Thirty-four measurements of elemental carbon were taken at Khobar and Camp Thunderock during the Gulf War by USAEHA. The average elemental carbon concentration at Khobar was 0.0115 mg/m^3, with a low measurement of 0.0009 mg/m^3 and a high measurement of 0.0306 mg/m^3. At Camp Thunderock, the average elemental carbon concentration was 0.025 mg/m^3 with a low measurement of 0.0017 mg/m^3 and a high measurement of 0.0538 mg/m^3.[86] The highest actual elemental carbon (soot) measurement is 23× below the adjusted OSHA PEL for carbon black.

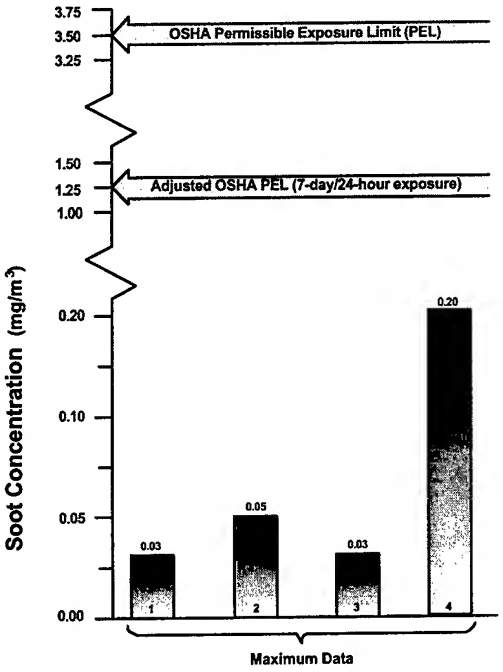

Figure 4. Maximum elemental carbon sample concentration measurements from 1–Kohbar, 2–Camp Thunderock, 3–RAND, and 4–USAEHA. Sample data are compared with the OSHA carbon black PEL and the adjusted OSHA carbon black PEL.

[85] Spektor, D.M.: "A Review of the Scientific Literature as it Pertains to Gulf War Illness — Oil Well Fires," *RAND*, Vol. 6 (1998). p. 16, Tables 2–5.

[86] USAEHA: "Final Report–Kuwaiti Oil Fire Health Risk Assessment (No. 39–26–1192–91, 5 May–3 December 1991), Appendix G — Sand and Ambient Air Sample Analysis, Vol. 1. Table G-14.

In a worst-case scenario, if the assumption that 23% of the PM_{10} reported by USAEHA is soot,[87] the following values result from a 153-day average exposure in the Gulf War Theater using summarized USAEHA PM_{10} data spanning the range between the minimum (Khobar) and maximum (U.S. Embassy) values (see Figure 4):[88]

- The 95% UCL for soot concentration ranges from 0.05 mg/m^3 to 0.20 mg/m^3.
- The mean soot concentration ranges from 0.03 mg/m^3 to 0.07 mg/m^3.

Applying the formula in Section III of this paper (Methodology) to estimate a 24-hour/7-day/week cumulative exposure for the 153-day duration, we find that our adjusted OSHA PEL for carbon black cumulative exposure equals 2.2 mg/m3 × years (see Figure 5).

- The 95% UCL for soot cumulative exposure ranges from 0.08 mg/m^3 × years to 0.35 mg/m^3 × years.
- The mean soot cumulative exposure ranges from 0.05 mg/m3 × years to 0.12 mg/m^3 × years.

The exposure of Gulf War veterans based on USAEHA estimates do not approach or exceed the adjusted OSHA PEL for carbon black. The highest estimated worst-case 95% cumulative soot exposure is nearly seven times below the adjusted OSHA PEL for carbon black, and the highest estimated worst-case mean (0.12 mg/m^3 × years) is nearly 19 times below the adjusted OSHA PEL for carbon black. The highest actual measurement for elemental carbon (0.0538 mg/m^3 at Camp Thunderock) was close to four times below the highest estimated soot concentration of 0.20 mg/m^3 presented above. It is worth noting that of 34 PM_{10} samples measured by USAEHA for elemental carbon (soot), only two samples equaled or exceeded 23% soot, six samples exceeded 10% soot, and the rest were below 10% soot.[89]

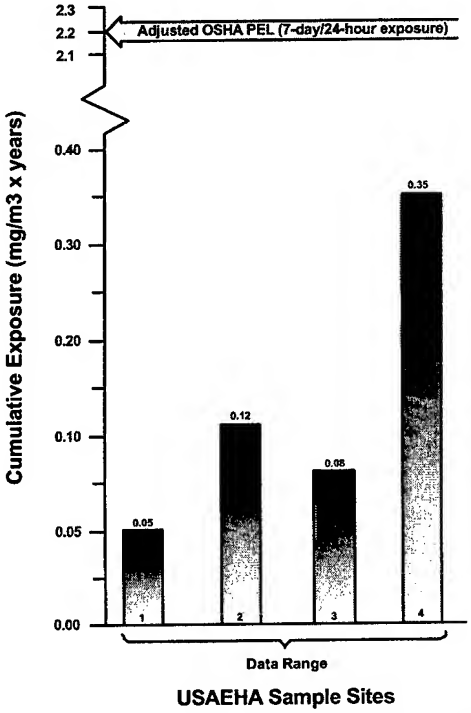

Figure 5. All cumulative exposure sample data ranges are from the USAEHA report (1, 2, 3, 4). Mean and maximum 95% cumulative soot exposure vs. cumulative exposure for a 24-hour, 7-day/week adjusted OSHA carbon black PEL.

Additional studies were considered to estimate various types of pollutants produced as a result of the Kuwaiti oil fires. As with the

[87] Spektor, D.M.: "A Review of the Scientific Literature as it Pertains to Gulf War Illness — Oil Well Fires," *RAND*, Vol. 6 (1998).

[88] [88] USAEHA: "Final Report–Kuwaiti Oil Fire Health Risk Assessment (No. 39–26–1192–91, 5 May–3 December 1991), Appendix G — Sand and Ambient Air Sample Analysis, Vol. 1. p. B-14 (Tables B-7 and B-8).

[89] USAEHA: "Final Report–Kuwaiti Oil Fire Health Risk Assessment (No. 39–26–1192–91, 5 May–3 December 1991), Appendix G — Sand and Ambient Air Sample Analysis, Vol. 1. Table G-2-18.

USAEHA studies, these papers present comparative evaluation of air quality during and after the Gulf War.

These studies derive estimates from sampling between May and September of 1991. The TSP matter concentration varied from 0.100 mg/m^3 to 0.935 mg/m^3, with average concentrations of 0.326 mg/m^3. Inhaled particulate matter at most locations in Kuwait and eastern Saudi Arabia was greater than air quality standards established by the Meteorology and Environmental Protection Administration (MEPA) and the EPA (see Table 3).[90]

The sampled PM_{10} concentration ranged between 0.238 mg/m^3 and 0.508 mg/m^3, with maximum daily values above 1.5 mg/m^3. The comparison indicates about a four-fold increase (or 77% average difference) in PM_{10} concentration levels. Every combusted metric ton of crude oil produces, on average, 20 kg of PM_{10}. This equates to 4–5 kg or an approximate 20%–25% soot component.[91,92] These analyses substantiate USAEHA data, which identify the PM_{10} soot fraction as approximately 23% or less.[93]

Table 3. Particulate matter air quality standards.

Particulate Matter (PM_{10}) Concentration (mg/m^3)	Averaging Time	Organization	Comments
0.340	1 day	MEPA	Not to exceed twice a month for TSP.
0.080	1 year	MEPA	Not to exceed once a year for TSP.
0.150	1 day	USEPA	USEPA standard is for PM_{10}.

Data analysis shows that the 95th percentile USAEHA PM_{10} data do not exceed the 1.25 mg/m^3 adjusted OSHA PEL for continual cumulative exposure to carbon black. Reasonable and allowable occupational or lifetime cumulative exposure levels were not approached.

If silica and carbon black exposures were considered additive, the combined soot and silica exposures for Gulf War soldiers would not exceed a combined additive level based on adjusted occupational threshold limits. As stated earlier, the effects of these dusts are significantly different on the lung. We do not believe such an additive analysis is scientifically justified.

The carbon black analysis is based on the assumption that the soot from the Kuwaiti oil fires is a well-combusted carbon-based material, similar in properties to commercial carbon black. High efficiency oil combustion during the Gulf War fires produced low VOC and PAH levels. VOC and PAH concentrations were lower in Gulf War regions when compared with levels found in most U.S. urban areas, and they were below recommended exposure levels.[94] In addition, the maximum inhaled level of soot

[90] Husain, T.: "Terrestrial and Atmospheric Environment during and after the Gulf War." *Environment International 24(1/2)*:189-196 (1997).
[91] Husain, T.: "Kuwaiti Oil Fires — Particulate Monitoring." *Atmos. Environ. 28(13)*:2235-2248 (1994).
[92] Husain, T.: "Kuwaiti Oil Fires — Source Estimates and Plume Characterization." *Atmos. Environ. 28(13)*:2149-2158 (1994).
[93] Ferek, R.J., Hobbs, P.V., Herring, J.A., Larsen, K.A., & Weiss, R.A.: "Chemical Composition of Emissions from the Kuwaiti Oil Fires," *J. Geophys. Res. 97(D13)14*:483-489 (1992).
[94] Spektor, D.M.: "A Review of the Scientific Literature as it Pertains to Gulf War Illness — Oil Well Fires," *RAND*, Vol. 6 (1998).

based on a worst-case, health-conservative assumption (considering the sooty material present during the Gulf War had toxicological properties most similar to carbon black) did not approach levels of significant health concern. The authors recognize the significant difference between much lower EPA standards for PM_{10} and those discussed here. The comparison shown here for Gulf War soot and carbon black is a reasonable comparison for similar, but by no means identical, materials. We, therefore, focused our analysis on the 73% fraction of the sand-based material that constituted the majority of particulate exposure to Gulf War veterans.

2. Iron Species

When inhaled, combined silica and iron species augment pulmonary reactions. As noted above, fracturing silica produces hydroxyl radicals. The hydroxyl radical production is associated directly with cellular damage. Iron-contaminated silica proves to be more pathological than inhalation of pure silica particles. A laboratory study using rats showed a 57% increased production of reactive species when compared to silica with low to no iron contamination.[95]

The evidence from these studies suggests a direct correlation between radical production and cytotoxicity. Thus, decreased radical generation leads to reduced cellular damage. Further, a direct relationship exists between radical production and cytotoxicity and the potency of the silica. Iron enhances the radical production in freshly fractured silica.

These studies also suggest that inhalation of quartz with high iron contamination causes significantly more damage to the alveolar air-blood barrier than an equal exposure to quartz with low iron contamination.[96] Investigators found that freshly milled silica contaminated with high iron was generally more inflammatory than milled silica with low iron contamination, as judged by the infiltration of leukocytes into the lung airspace.

3. Organic Material

Inhalation of airborne microorganisms associated with organic dusts may alter respiratory function and activate lung cells, including alveolar macrophages. This interaction promotes hypersensitivity reaction in the lungs. Microorganisms and their metabolic products are considered among the most pathogenic compounds associated with organic dusts.[97] Activated lung alveolar macrophages produce reactive oxygen intermediates that contribute to tissue inflammation. These reactive oxygen intermediates

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[95] Vallyathan, V., Leonard, S., Kuppusamy, P., Pack, D., Chzhan, M., Sanders, S.P., & Zweir, J.L.: "Oxidative Stress in Silicosis: Evidence for the Enhanced Clearance of Free Radicals from Whole Lungs." *Molecular and Cellular Biochemistry 168:*125-132 (1997).

[96] Castranova, V., Vallyathan, V., Ramsey, D.M., McLaurin, J.L., Pack, D., Leonard, S., Barger, M.W., Ma, J.Y.C., Dalal, N.S., & Teass, A.: "Augmentation of Pulmonary Reactions to Quartz Inhalation by Trace Amounts of Iron-Containing Particles." *Environ. Health Report 105 (Suppl. 5):*1319-1324 (1997).

[97] Jacobs, R.R.: "Airborne Endotoxins: An Association with Occupational Lung Disease." *J. Appl. Ind. Hyg. 4:*50-56 (1989).

also are capable of destroying many cellular structure elements as well as act as chemoattractants and initiate pulmonary inflammation.[98]

An example of organic dust-induced pulmonary inflammation was observed and documented in the village of Al-Eskan, Saudi Arabia. Al-Eskan, in Riyadh, is one of several villages established by King Khalid. During Gulf War operations, U.S. forces moved into these apartment-type dwellings. A human population had never occupied this compound. In order to make these living quarters habitable, the soldiers implemented a cleaning effort that resulted in large quantities of respirable dust — particulate that was a mixture of fine Saudi sand and pigeon droppings laden with many allergeric substances, including spores, pollen, fungi, and other microorganisms.

Occupying Al-Eskan abruptly exposed a significant number of soldiers to unique environmental hazards. These mixtures of fine organic particulate and pigeon droppings triggered a "Phase I" hypersensitive allergic response. A "Phase I" response is aggravated by the various organic pathogens and develops into an opportunistic lung infection. "Phase II" develops either in continuation with persistent symptoms or evolves after a few months into complications associated with impaired respiratory function. These site-specific exposures resulted in an acute lung condition diagnosed as "Al-Eskan Disease" or "Desert Storm Pneumonitis."[99]

This study is considered a cluster event and not representative of ambient particulate exposures experienced by the majority of Gulf War soldiers. Al-Eskan provides unique health data relating to the combined exposure effects. Additional controlled re-examination of this exposure situation might lead to better understanding of this and related ailments for future deployment of troops in desert environments.

4. Tobacco Smoke

Combined effects of exposure to respirable free silica concentrations and tobacco smoke produce a significant dose–response relationship between lung disease, silica dust particle-years, and cigarette equivalent pack-years. A mortality study of gold miners identifies an increased relative risk for lung disease when silica dust exposures and smoking lifestyles are considered. The multiplicative model better fits a combined effect than the simple additive model.[100]

Although a correlation is established between silica dust inhalation and silicosis, careful consideration must be given to the concentration of free crystalline silica. Primary studies investigating this association identify dusts containing a high level of free

[98] Milanowski, J.: "Effect of Inhalation of Organic Dust-Derived Microbial Agents on the Pulmonary Phagocytic Oxidative Metabolism of Guinea Pigs." *J. Toxicol. Environ. Health 53* (Part A):5-18 (1998).
[99] Al-Frayh A.S., Korenyi-Both, A.L., & Korenyi-Both, A.L.: "Al-Eskan Disease: Desert Storm New Data and Round-up Pneumonitis: New Data and Round-up." Department of Pediatrics, College of Medicine, King Saudi University, Riyadh, Saudi Arabia, *Vol. 8(1)*:1-8 (1997).
[100] Hnizdo, E., & Sluis-Cremer, G.K.: "Silica Exposure, Silicosis, and Lung Cancer: A Mortality Study of South African Gold Miners." *Brit. J. Ind. Med. 48*:53-60 (1991).

crystalline silica at concentrations greater than 30%.[101] Gulf War soldiers were exposed to respirable crystalline silica concentrations of 6.5% or less. This difference would account for a decreased risk of lung disease resulting from the combined effect of silica dust exposure and tobacco smoking.

VII. RISK CHARACTERIZATION

Studies conducted on laboratory animals and humans indicate that fibrogenic and silicotic lung lesions result from the inhalation of crystalline silica. Lung cancer is a secondary function to formation of these lesions.[102] Epidemiological studies supporting the increased risk of lung cancer from silica exposure are summarized below.

A cohort mortality study was administered for 2,342 male workers exposed to crystalline silica, predominantly cristobalite, in a diatomaceous earth mining and processing facility in California. Between 1942 and 1994, mortality excesses were detected for nonmalignant respiratory diseases (NMRDs) (standardized mortality ratio [SMR] = 2.01, 95% confidence interval [CI] = 1.56–2.55) and lung cancer (SMR = 1.29, 95% CI = 1.01–1.61). NMRD mortality rose sharply with cumulative exposure to respirable crystalline silica. The rate ratio for lung cancer reached 2.15 (95% CI = 1.08–4.28) in the highest exposure category. These associations were unlikely to have been confounded by smoking or asbestos exposure. The findings indicate a strong dose–response relationship for crystalline silica and NMRD mortality.[103]

The International Agency for Research for Cancer (IARC) has judged that evidence for the carcinogenicity of silica in animals is sufficient and for humans it is limited. Animal findings are based on rat inhalation studies. Because of the many uncertainties involved in extrapolating to humans from animal data, more rational risk assessments are achieved when data from silica-exposed workers are used rather than laboratory animal findings.[104]

Occupations characterized by high exposure to crystalline silica result in excessive rates of lung cancer mortality. Respiratory cancer excesses are reported from North America and Europe for the following dusty trades: iron and steel foundry workers, steel carting workers, sand blasters, metal molders, uranium miners, and ceramic workers. These findings are reinforced by two reports from the Swedish Pneumoconiosis Register and the Ontario Ministry of Labour. Based on this evidence, silicosis provides statistically significant risks of lung cancer mortality in animal studies, and silica can be

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[101] Hnizdo, E.: "Health Risks among White South African Gold Miners — Dusts, Smoking and Chronic Obstructive Pulmonary Disease." *S. Air Med. J. 81:*512-517 (1992).

[102] Klein, A.K., & Christopher, J.P.: "Evaluation of Crystalline Silica as a Threshold Carcinogen." *Scand. J. Work Environ. Health 21 (Suppl. 2)*:95-98 (1995).

[103] Klein, A.K., & Christopher, J.P.: "Evaluation of Crystalline Silica as a Threshold Carcinogen." *Scand. J. Work Environ. Health 21 (Suppl. 2)*:95-98 (1995).

[104] World Health Organization: "IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Silica, Some Silicates, Coal Dust and para-Aramid Fibrils." *IARC Monographs 68:*203-211 (1997).

an initiating carcinogen or can act as a co-carcinogen initiator when combined with benzo(a)pyrene.[105]

A cohort mortality study was conducted of white men employed for at least one year between 1939 and 1966 at three plants of a single U.S. company. This was designed to evaluate the risk of lung cancer and nonmalignant respiratory disease among workers exposed to respirable silica and nonfibrous (nonasbestiform) talc dusts in the manufacture of ceramic plumbing fixtures. A follow-up study of 2,055 men through January 1, 1961, indicated a substantial excess of nonmalignant respiratory disease among those with high levels of exposure to respirable silica dust (SMR = 2.26). No significant increases in lung cancer risks were noted.[106]

Evidence as to the causal relationship between silica exposure, silicosis, and carcinogenic risks are growing. Silica exposed workers consistently demonstrate elevated relative risk values for lung cancer. Although inconclusive, when exposures are adjusted for smoking, relative risks are shown to increase with silica dose. It is also understood that human carcinogenicity is not detected in all industrial circumstances and may be significantly dependent on inherent characteristics of the crystalline silica. This evidence contributes to a strong understanding of the mechanistic events leading from quartz deposition, silicosis, and cancer. Reported hazards posed by quartz species are not consistent. Risks and probable carcinogenic effects exist but may vary dramatically depending on the silica sample origin, its interaction with other chemicals/minerals, and circumstance of human exposure.

VIII. FINDINGS AND CONCLUSIONS

This study identified that TSP and respirable particulate matter exposure levels were often exceeded. In addition, the respirable silica fraction was identified as the greatest health concern.

A literature review indicated negligible to nonexistent health risk from other inhaled particulate material during the Gulf War. However, because silica is the most toxic component of desert dust, the emphasis of this report is on the potential health effects of respirable silica exposure in troops.

Respirable dust particulates have been associated with increases in mortality and risks of respiratory and cardiovascular disease. Further, exposures to higher concentrations of respirable dust particulates have been associated with changes in lung function, damage to lung tissue, and altered respiratory defense mechanisms. Occupational exposures to respirable silica have produced the same types of changes in lung tissue and functions. However, depending on the extent of exposure, these changes may be more pronounced and severe.

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[105] Goldsmith, D.F., Guidotti, T.L., & Johnston, D.R.: "Does Occupational Exposure to Silica Cause Lung Cancer?" *Am. J. Ind. Med. 3:*423-440 (1982).

[106] Thomas, T.L., & Stewart, P.A.: Mortality from Lung Cancer and Respiratory Disease among Pottery Workers Exposed to Silica and Talc." *Am. J. Epidemiol. 125:*35-43 (1987).

Silicotic disease is enhanced when the majority of respired silica is freshly fractured, such as in a workplace setting where stone surfaces are ground, but not in a natural setting such as the Gulf War or typical desert exposure. Natural silica exposure at high levels for long periods can produce disease, but new evidence indicates that freshly fractured man-made exposure is far more harmful in acute settings.

Studies in laboratory animals and humans indicate that fibrogenic scarring and silicotic lung lesions are the primary pathological result from the crystalline silica inhalation. Lung cancer is a secondary pathological result to the formation of these lesions. Epidemiological studies show small increased relative risks of cancer in workers exposed over many years to crystalline silica.

The 1991 military analysis of respirable desert dust inhaled by soldiers in the Gulf War showed it to contain an average of 4.3% and a maximum of 6.5% silica. This literature review indicated that these values are typical for respirable desert dusts.

Health conservative assumptions indicate a NOAEL to be 20 times the predicted exposure scenarios. A lifetime NOAEL level was shown to be eight times the worst-case dose and cumulative exposure scenario developed for Gulf War soldiers.[107]

The average cumulative respirable silica exposure and dosage for Gulf War soldiers was roughly 1/50th to 1/100th the 1 mg/m^3 × year NOAEL. This equates into a 50- to 100-fold margin of safety for a no observed adverse effect level (Tab B: Figures 6–9).

Based on the scientific literature and human exposure calculations, this study concludes that adverse health effect risks from respirable silica dusts are well below health concern levels. The risk of silicosis associated with ambient levels of respirable silica is estimated by first adjusting from 8-hour occupational exposure equivalents. This method is consistent with EPA dose–response assessments.[108] Chronic health effects are not expected given that average cumulative exposure is below current workplace adjusted guidelines. Based on OSHA or NIOSH limits for silica adjusted for evaluation of Gulf War soldiers' round-the-clock exposure, the dosages are an order of magnitude and the cumulative exposure two-orders of magnitude below the ambient NOAEL. Neither acute nor chronic effects are expected given that average cumulative exposure is below current workplace guidelines adjusted for continual 24-hour exposures.

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[107] U.S. Army: "Final, Kuwait Oil Fire Health Risk Assessment — Appendix B." U.S. Army Environmental Hygiene Agency, Aberdeen Proving Ground, MD, 1994.

[108] U.S. EPA/Office of Research and Development: *Ambient Levels and Noncancer Health Effects of Inhaled Crystalline and Amorphous Silica: Health Issue Assessment* (EPA/600/R–95/115). November 1996. pp. 7-1–7-6.

IX. RECOMMENDATIONS FOR FURTHER STUDY

Several areas of research could improve the understanding of the potential long-term health problems in soldiers exposed to respirable desert dust and other pollutants. Findings from this research could be used as an addendum to this report and factored into the current health assessment. Six of the most important recommendations for further study are described below.

- *Design and execute a study to examine respirable particulate from the Gulf War region for surface iron, aluminum, and free-radical surface activity.* The study could consist of samples collected for total suspended particulate and respirable dust (PM_{10} and/or industrial hygiene respirable breathing zone particulate collected with a cyclone) under ambient and sandstorm conditions and analyzed within a required, consistent short time period by Electron Spin Resonance (ESR) spectroscopy.

- *Design and execute a study to examine respirable particulate from the Gulf War region for surface iron, aluminum and free radical surface activity under high wind conditions.* The types of samples and analysis would be the same as above. The value would be experimental control of the soil and weather conditions to estimate the fresh silica fractionation and potential biological activity of silica subjected to high wind conditions.

- *Analyze a subset of the 1991 Gulf War PM_{10} industrial hygiene and bulk soil samples using X-ray Diffraction (XRD) specifically for crystalline silica.* The average and maximum values used for the exposure assessment are based on the silicon-rich portion of material from the USAEHA 1991 report, which may have overstated the concentration of crystalline silica in the samples. Analysis would confirm the 100% concentration of crystalline silica used in this exposure estimate. Conversations with analysts of the samples from the monitoring studies indicate that it is highly unlikely that more alpha-quartz is present than the silicon-rich fraction analyzed in the 1991 samples. Thus, health risk estimates would not likely increase. If upon reanalysis the concentration of crystalline silica were significantly less than the silicon-rich "quartz" percentage originally reported in the 1991 USAEHA report, health risk estimates would decrease.

- *Re-examine the Gulf War soldiers' dust exposure data in light of new $PM_{2.5}$ standards that are emergent from EPA.* The EPA might promulgate standards for this particulate in the range of 15 $\mu g/m^3$ to 65 $\mu g/m^3$. These levels would have been routinely exceeded from the Gulf War data reviewed. A closer examination of new or different health effects from exposure may be useful when these standards are finalized. Monitoring samples collected by USAEHA during the Gulf War would allow such a detailed examination.

- *Conduct short-term personal industrial hygiene monitoring and ESR examination for silica and other dusts in certain dusty jobs such as marching behind tanks operating in the desert.* This could provide a real-world, worst-

case evaluation of the freshly fractured nature of silica in the Gulf environment and evaluate the need for better work practices to reduce short-term dust exposure levels.

- *Examine further the need for more chemical comparison of Kuwaiti oil fire soot and other carbon black (soot) for similarities and differences in order to better substantiate scientifically any health risk from exposure.* Current evidence indicates similarities to carbon black commonly manufactured as a fossil fuel combustion product. Further chemical analysis would verify or disprove this assumption.

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TAB B — Detailed Exposure Estimates

Figure 6. Respirable silica exposure for Gulf War veterans is identified and compared with occupational health effects studies. Cumulative exposure (mg/m^3 × years) levels are calculated from USAEHA air analysis data collected at the following sample sites: 1–KKMC, 2–Khobar, 3–Riyadh (Eskan), 4–Jubayl, 5–Camp Thunderock, 6–U.S. Embassy, 7–Military Hospital (USAEHA, 1991 & 1994). Air analysis samples are respirable particulate matter and estimated to contain an average concentration of 4.3% silica quartz. A no observed adverse effect level (NOAEL) identifies the point at or below which human health risks are not expected to occur.

Figure 7. Respirable silica exposure for Gulf War veterans is identified and compared with occupational health effects studies. Cumulative exposure (mg/m^3 × years) levels are calculated from USAEHA air analysis data collected at the following sample sites: 1–KKMC, 2–Khobar, 3–Riyadh (Eskan), 4–Jubayl, 5–Camp Thunderock, 6–U.S. Embassy, 7–Military Hospital (USAEHA, 1991 & 1994). Air analysis samples are respirable particulate matter and estimated to contain an average concentration of 6.5% silica quartz. A no observed adverse effect level (NOAEL) identifies the point at or below which human health risks are not expected to occur.

Figure 8. Respirable silica exposure for Gulf War veterans is identified and compared with occupational health effects studies. Total respirable silica dose (mg) levels are calculated from USAEHA air analysis data collected at the following sample sites: 1–KKMC, 2–Khobar, 3–Riyadh (Eskan), 4–Jubayl, 5–Camp Thunderock, 6–U.S. Embassy, 7–Military Hospital (USAEHA, 1991 & 1994). Air analysis samples are respirable particulate matter and estimated to contain an average concentration of 4.3% silica quartz. A no observed adverse effect level (NOAEL) identifies the point at or below which human health risks are not expected to occur.

Figure 9. Respirable silica exposure for Gulf War veterans is identified and compared with occupational health effects studies. Total respirable silica dose (mg) levels are calculated from USAEHA air analysis data collected at the following sample sites: 1–KKMC, 2–Khobar, 3–Riyadh (Eskan), 4–Jubayl, 5–Camp Thunderock, 6–U.S. Embassy, 7–Military Hospital (USAEHA, 1991 & 1994). Air analysis samples are respirable particulate matter and estimated to contain an average concentration of 6.5% silica quartz. A no observed adverse effect level (NOAEL) identifies the point at or below which human health risks are not expected to occur.

Silicosis: 48% cumulative risk and 30 years exposure from high range estimated silica concentrations, Gift_1997, 352 & Caviarri_1995

Silicosis: 48% cumulative risk and 30 years exposure from low range estimated silica concentrations, Gift_1997, 352 & Caviarri_1995

Silicosis: 77% cumulative risk and 35 years of exposure, HnizdoE_1993, 454

Progressive Massive Fibrosis (PMF): 7 cases or 11% of study groups and 1-9 years high silica dust exposures, BanksDE_1981, 447

Silicosis: 25% cumulative risk and 25 years exposure, HnizdoE_1993, 453-456

Chronic Obstructive Pulmonary Disease (COPD): 24 years exposure, HnizdoE_1992, 513

Pneumoconiosis category I: 30% cumulative risk and 46 years exposure, Muir_1989, 37

Silicosis: evidenced by 24% of radiographs and 17 years exposure from Hong Kong granite workers, Gift_1997, 352 & Ng & Chan_1994

Occupational Health Effects Range

No Observed Adverse Effect Level (NOAEL)

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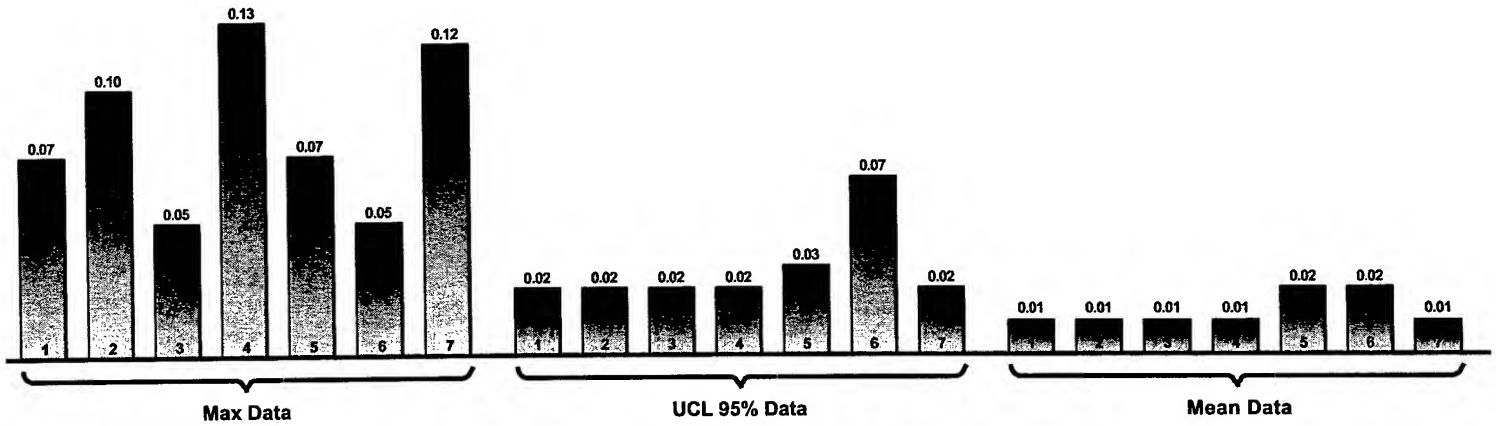

Figure 6: USAEHA Sample Sites

Silicosis: 48% cumulative risk and 30 years exposure from high range estimated silica concentrations, Gift_1997, 352 & Caviarri_1995

Silicosis: 48% cumulative risk and 30 years exposure from low range estimated silica concentrations, Gift_1997, 352 & Caviarri_1995

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Occupational Health Effects Range

No Observed Adverse Effect Level (NOAEL)

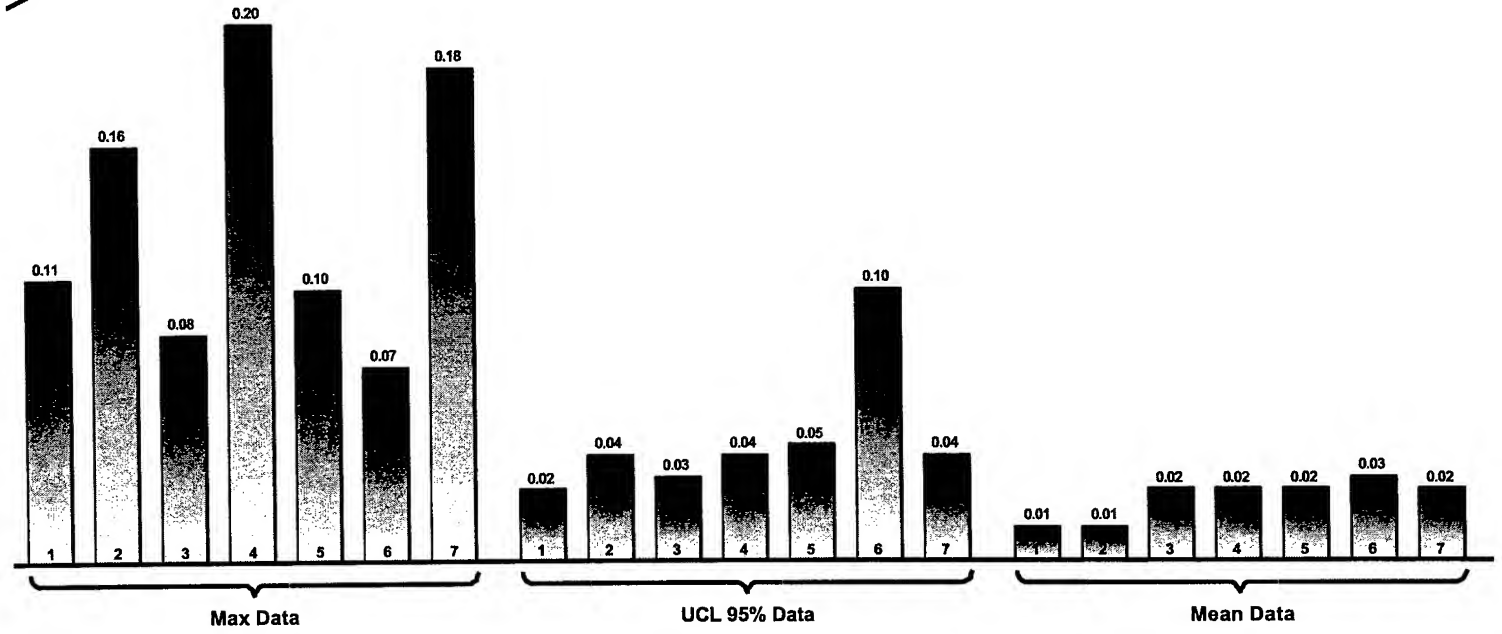

Figure 7: USAEHA Sample Sites

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Occupational Health Effects Range

No Observed Adverse Effect Level (NOAEL)

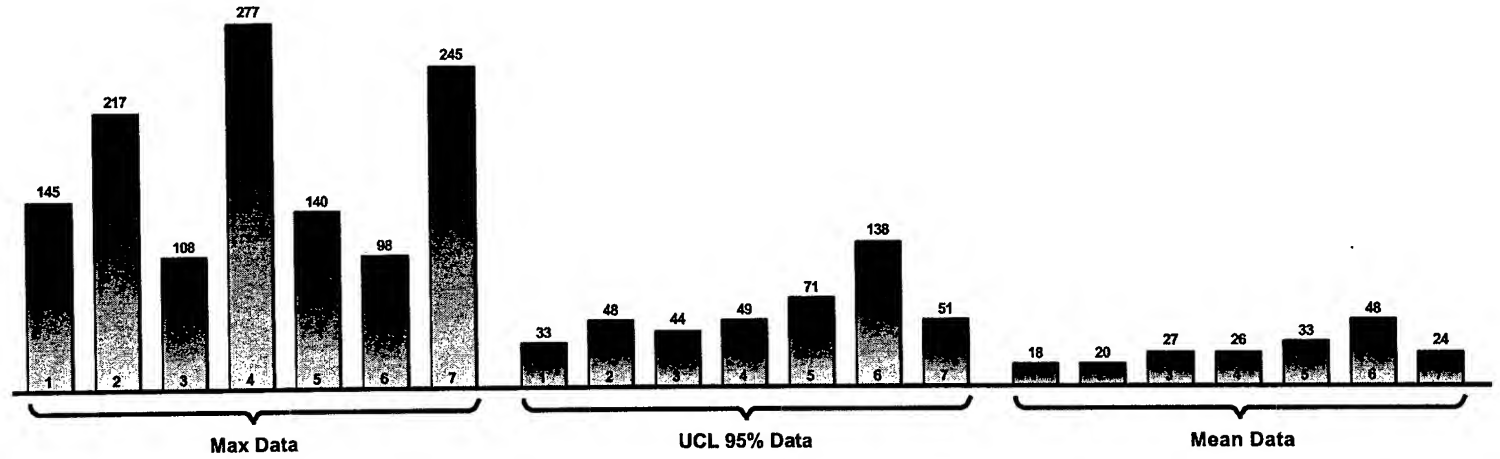

Figure 8: USAEHA Sample Sites

Silicosis: 48% cumulative risk and 30 years exposure from high range estimated silica concentrations, Gift_1997, 352 & Caviarri_1995

Silicosis: 48% cumulative risk and 30 years exposure from low range estimated silica concentrations, Gift_1997, 352 & Caviarri_1995

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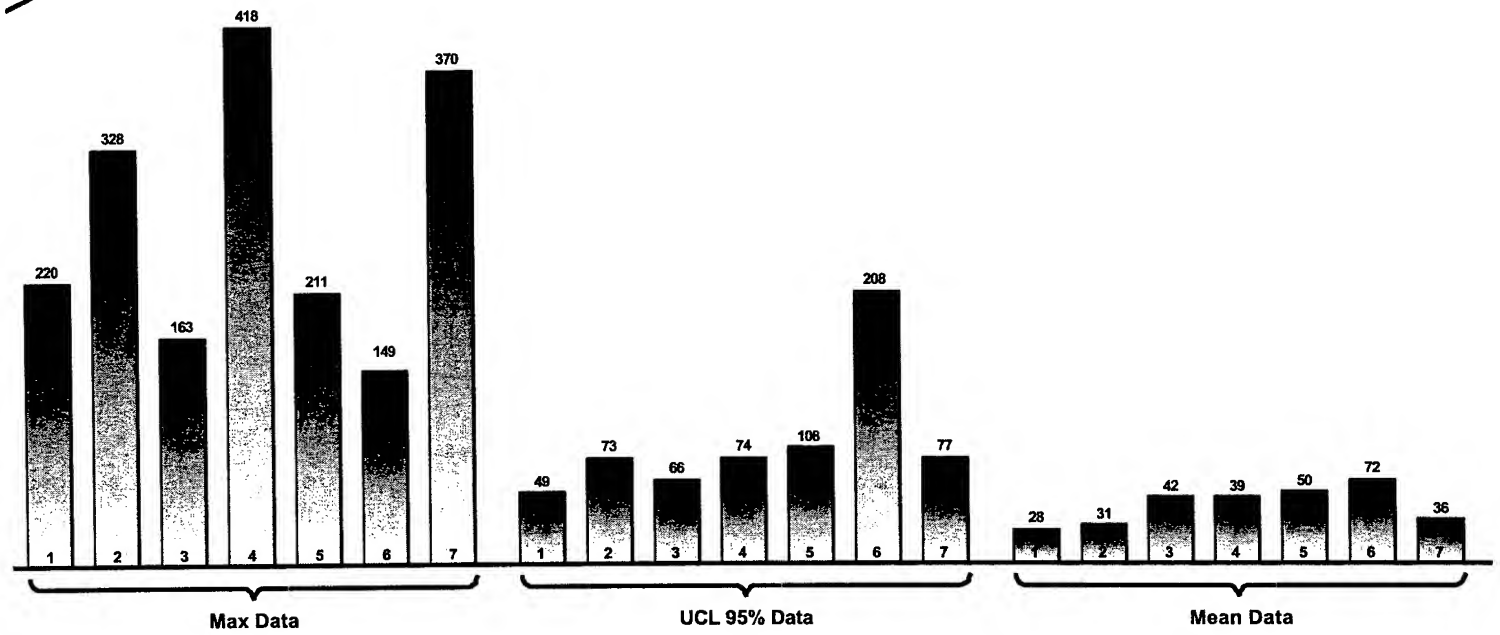

Figure 9: USAEHA Sample Sites

TAB C — Acronyms

ACGIH.....American Conference of Governmental Industrial Hygienists
BAL.....Bronchalveolar Lavage
BTEX..... Benzene, Toluene, Ethyl Benzene, and Xylene
CHPPM.....U.S. Army Center for Health Promotion and Preventive Medicine
DOD.....U.S. Department of Defense
EPA..... U.S. Environmental Protection Agency
ESR.....Electron Spin Resonance
IARC.....International Agency for Research for Cancer
KMPH.....Kuwait Ministry for Public Health
LDH..... Lactate Dehydrogenase
MSHA.....Mine Safety and Health Administration
NAAQS.....National Ambient Air Quality Standards
NIOSH.....National Institute for Occupational Safety and Health
NMRD.....Nonmalignant Respiratory Disease
NOAEL.....No Observed Adverse Effect Level
OSHA.....Occupational Safety and Health Administration
PAHs..... Polycyclic Aromatic Hydrocarbons
PEL.....Permissible Exposure Limit
PM_{10}..... Particulate Matter at or below 10 microns
RBC.....Red Blood Cells
REL.....Recommended Exposure Limit
TLVs® .....Threshold Limit Values® (ACGIH)
TSP..... Total Suspended Particulate
USAEHA.....U.S. Army Environmental Hygiene Agency
VOCs.....Volatile Organic Compounds

TAB D — Glossary

Acute: 1. Referring to a health effect — brief; not chronic; sometimes loosely used to mean severe. 2. Referring to exposure — brief, intense, short-term; sometimes specifically referring to a brief exposure of high intensity.

Aerodynamic Equivalent Diameter (AED): The settling rate of suspended particles and their penetration into the respiratory tract is in accordance with the particle AED, an expression that accounts for the inertial and aerodynamic drag properties of particles. The AED depends on the particle density, shape, and size. The particle AED is defined as the diameter of a smooth, unit density (ρ_o = 1 gram per cubic centimeter [g/cm^3]) sphere having the same terminal settling velocity as the actual particle. The use of the AED enables one to standardize particles of different shapes, smoothness, and densities for direct comparative purposes.

Alpha-quartz: The most stable form of crystalline silica in the environment. The vast majority of natural crystalline is in the form of alpha-quartz.

Alveolar Phagocytes: Rounded granular phagocytic cells within the alveoli of the lungs, which ingest inhaled material (*see* Macrophage).

Ambient: Surrounding, encompassing; pertaining to the environment in which an organism or apparatus functions.

Anthropogenic: Concerning human activities or of human origin.

Asthma: A condition of the lungs in which there is widespread narrowing of airways, varying over short periods of time either spontaneously or as a result of treatment, due in varying degrees to contraction (spasm) of smooth muscle, edema of the mucosa, and mucus in the lumen of the bronchi and bronchioles; these changes are caused by the local release of spasmogens and vasoactive substances (e.g., histamine, or certain leukotrienes or prostaglandin) in the course of an allergic process.

Biological Reactivity: Pertains to the interaction of a material with living tissues and cells. For example: DNA-damaging activity of amorphous, crystalline, and other silica species.

Bronchoalveolar Lavage:

Irrigation or washing of the bronchoalveolar ducts and terminal bronchioles.

Chronic: 1. Referring to a health-related state, lasting a long time. 2. Referring to exposure, prolonged or long-term, sometimes also meaning low-intensity. 3. The U.S. National Center for Health Statistics defines a chronic condition as one of three months' duration or longer.

Chronic Lymphocytic Alveolitis:
Alveolitis — inflammation of alveoli; lymphocytic — pertaining to lymphocytes; lymphocyte — a white blood cell formed in lymphatic tissue throughout the body (e.g., lymph nodes, spleen, thymus, tonsils, Peyer's patches, and sometimes in bone marrow) and in normal adults comprising approximately 22%–28% of the total number of leukocytes in the circulating blood.

Chronic Obstructive Pulmonary Disease (COPD):
Characterized by the irreversible (though sometimes variable) obstruction of the lung airways; includes chronic bronchitis and emphysema. Also known as Obstructive Airway Disorder.

Cohort Mortality Study:
A follow-up epidemiological study where information about the exposure status to a disease suspect or causing agents in the population studied is known and compares disease or death of a population of exposed and unexposed subjects.

Confidence Interval (CI):
Statistically determined upper and lower bound 95% chance that a measurement will occur within a set of measurements or values.

Crystalline Silica: *See* quartz.

Cumulative Exposure:
1. Proximity and/or contact with a source of a disease agent that accumulates or piles up in such a manner that effective transmission of the agent or harmful effects of the agent may occur.

Cumulative (Total) Dosage:
Total amount of a material or agent to which an organism is exposed for a period of time.

Cytolytic: Pertaining to, characterized by, or causing the dissolution or destruction of cells.

Cytotoxic: Detrimental or destructive to cells; pertaining to the effect of an noncytophilic antibody on a specific antigen, frequently, but not always, mediating the action of complement.

Dose: Intake in unit of mass (mg) calculated or estimated that is taken into the body. In this review, 100% of inhaled intake is assumed absorbed by the body.

Dyspnea: Shortness of breath, a subjective difficulty or distress in breathing, usually associated with disease of the heart or lungs; occurs normally during intense physical exertion or at high altitudes.

Edema: An increase in permeability leading to the extravasion of plasma proteins and fluid, resulting in localized swelling of tissues.

Endothelial: Pertaining to, or made up of, the surface cells that line the internal cavities of organs.

Erythrocytes: Mature red blood cells.

Fibrogenic: Conducive to the development of fibers or fibrosis.

Fibrotic Lung Disease:
Formation of fibrous tissue as a reparative or reactive process — as opposed to formation of fibrous tissue as a normal constituent of an organ or tissue — causing organ impairment, dysfunction, and disease.

Homeostasis: A tendency to be stable in the normal state (internal environment) of an organism.

Inflammatory Response:
A response pertaining to, characterized by, causing, resulting from, or becoming affected by inflammation. Inflammation is a fundamental pathological process consisting of a dynamic complex of cytological and chemical reactions that occur in the affected blood vessels and adjacent tissues in response to an injury or abnormal stimulation caused by a physical, chemical, or biological agent.

Inflammatory Cells (Netrophils and Lymphocytes):
Neutrophil, a granular leukocyte having a nucleus with three to five lobes connected by slender threads of chromatic, and cytoplasm containing fine, inconspicuous granules. Lymphocyte is a mononuclear leukocyte, 7 μm to 20 μm in diameter, with a deeply staining nucleus containing dense chromatin and pale blue staining cytoplasm.

Macrophage (Alveolar):
Mononuclear cells within the lung tissues that are largely scavengers, ingesting dead tissue and degenerated cells.

Margin of Safety: The ratio of the no observed adverse effect level (NOAEL) to actual exposures.

Maximum: The greatest observation or measurement. The highest degree or point possible.

Mean: The arithmetic or numerical average as a calculation of central tendency in a frequency distribution.

Minimum: The lowest observation or measurement. The lowest degree or point possible.

Necrosis: Death of tissue, usually as individual cells, groups of cells, or in small-localized areas.

Neutrophil: A granular leukocyte having a nucleus with three to five lobes connected by slender threads of chromatic, and cytoplasm containing fine, inconspicuous granules.

NMRD: Nonmalignant Respiratory Disease(s) — pulmonary and respiratory tract diseases not having the properties of anaplasia, invasion, and metastasis (such as asthma or emphysema).

NOAEL: No Observed Adverse Effect Level — a toxicological reference level to a dose, cumulative exposure level, or time-weighted average (TWA) below which pathological consequences from exposure are not expected.

Obstructive Airway Disorders:
See Chronic Obstructive Pulmonary Disease (COPD).

Occupational: Arising from, or related to, the workplace.

Pathogenic: Giving origin to disease or morbid symptoms.

Particulate: Composed of separate tiny masses of material or particles.

Parts per Billion (ppb):
A chemical concentration term equivalent to 1 billionth of the whole; for example: 1 microgram per kilogram.

Peroxidation: The oxidation of organic substrates in the presence of hydrogen peroxide usually catalyzed by iron porphyrin enzymes.

Phagocyte: A cell possessing the property of ingesting bacteria, foreign particles, and other cells. Phagocytes are divided into two general classes: 1) microphages — polymorphonuclear leukocytes that ingest chiefly bacteria and foreign cells; and 2) macrophages — mononucleated cells (histiocytes and monocytes) that are largely scavengers, ingesting dead tissue, particulates, and degenerated cells.

Pleural Diseases: Diseases of the serous membrane investing the lungs and lining the thoracic cavity, completely enclosing a potential space known as the pleural cavity.

Pneumonitis: Inflammation of the lungs.

PM_{10}: Particulate matter 10 microns or less in size. Collected using a thoracic air sampler for particulate matter that meets the performance criteria specified by the U.S. EPA in 40 CFR Part 50.6 and 40 CFR Part 53. The performance criteria includes a collection efficiency of 100% for particles of 0–1 µm aerodynamic equivalent diameter (AED), 89.3% at 4 µm AED, 55.1% at 10 µm AED, 50.9% at 10.5 µm AED, 4.1% at 15 µm AED, and 0% at 16 µm AED. The performance criteria are such that this type of sampler is more like a thoracic air sampler than a respirable air sampler.

Pulmonary Phagocytes: Cells that ingest microorganisms or other cells and foreign particles within the lung.

Quartz: A form of hexagonal crystalline silica or silicon dioxide (SiO_2) occurring in abundance, most often in a colorless, transparent form but also sometimes in colored varieties used in semiprecious stones; the principal constituent of ordinary sand.

Radicals: A group of atoms that enter and exit a chemical combination without change, and that form the fundamental constituents of a molecule having a short half-life (10 -5 seconds or less in an aqueous solution) and carries an unpaired electron.

Rales: An abnormal respiratory sound heard in auscultation and indicating some pathological condition.

Respirable: The portion of an aerosol that is capable of entering the gas exchange regions of the lungs if inhaled. By convention, a particle-size-selective fraction of the total airborne dust with aerodynamic diameters less than approximately 10 µm that has a 50% deposition efficiency for particles with an aerodynamic diameter of approximately 4 µm. An ideal respirable sampler includes a

collection efficiency of 100% at 0 μm AED, 97% at 1 μm AED, 91% at 2 μm AED, 74% at 3 μm AED, 50% at 4 μm AED, 30% at 5 μm AED, 17% at 6 μm AED, 9% at 7 μm AED, 5% at 8 μm AED, and 1% at 10 μm AED.

Standardized Mortality Ratio (SMR):
The ratio of disease or death of an exposed population under study to a potential disease-causing agent to an unexposed population.

Thoracic: That portion of the respiratory tract that includes the lungs, both the conducting airways (tracheobronchial region) and the pulmonary region (alveolar region where gas exchange occurs). An ideal thoracic sampler includes a collection efficiency of 100% at 0 μm AED, 96% at 1 μm AED, 89% at 4 μm AED, 50% at 10 μm AED, 18% at 15 μm AED, 2% at 25 μm AED, 1% at 30 μm AED, and 0% at 40 μm AED. Particles that penetrate into the thoracic region will deposit either in the tracheobronchial region or the pulmonary region, depending on the particle AED and the collection efficiency of the respiratory tract for a given particle AED. If the thoracic size particles are small enough (about 10 μm AED or less), they may penetrate into the pulmonary region (alveolar region where gas exchange occurs), with particles smaller than 10 μm AED penetrating into the pulmonary region with greater efficiency.

TLVs®: Threshold Limit Values®, established by the American Conference of Governmental Industrial Hygienists (ACGIH), are voluntary occupational exposure limits to chemical and physical agents.

TSP: Total Suspended Particulate, referring to the entire range of ambient air matter that can be collected gravimetrically from the submicron level up to 50 μm in aerodynamic diameter.

95% Upper Confidence Limit (95% UCL):
The value that, when calculated repeatedly for random drawn subsets of data, equals or exceeds the true mean 95% of the time.